The Relevance of Different Patterns of Instability in the Treatment of Borderline Personality Disorder – Results of the Basel Borderline Inpatient Study (BABIS)

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Declaration

My cumulative dissertation is based on five manuscripts, of which four are already published and one is in preparation. All manuscripts are results of the Basel Borderline Inpatient Study (BABIS) which was founded by a research grant from the Swiss National Science Foundation (SNF).

I hereby declare that the present work is originally written by me without any unauthorized aid and that this work has not been and will not be submitted elsewhere for publication. Statement of others or citations are indicated. My contributions were substantial and independent in all five manuscripts, in collaboration with the co-authors mentioned in the articles.

Gremaud-Heitz, D., Stewart, J., Dammann, G. (2011). Konzept der atypischen Depression und deutsche Übersetzung der "Atypical Depression Diagnostic Scale (ADDS)". *Schweizer Archiv für Neurologie und Psychiatrie, 162(4):*148-154

Gremaud-Heitz, D., Riemenschneider, A., Walter, M., Sollberger, D. Küchenhoff, J., Dammann, G. (2014). Comorbid atypical depression in borderline personality disorder is common and correlated with anxiety-related psychopathology. *Comprehensive Psychiatry, 55:*650-656

Sollberger, D., Gremaud-Heitz, D., Riemenschneider, A., Küchenhoff, J., Dammann, G., Walter, M. (2012). Associations between Identity Diffusion, Axis II Disorder, and Psychopathology in Inpatients with Borderline Personality Disorder. *Psychopathology, 45:*15-21 DOI:10.1159/000325104

Sollberger, D., Gremaud-Heitz, D., Riemenschneider, A., Agarwalla, P., Benecke, C., Schwald, O., Küchenhoff, J., Walter, M., Dammann, G. (2014). Change in Identity Diffusion and Psychopathology in a Specialized Inpatient Treatment for Borderline Personality Disorder. *Clinical Psychology and Psychotherapy DOI: 10.1002/cpp.1915*

Dammann, G., Riemenschneider, A., Walter, M., Sollberger, D., Küchenhoff, J. , Gremaud-Heitz, D. (2015). The Impact of Interpersonal Problems in Borderline Personality Disorder Inpatients on Psychopathology and Treatment Outcome. *In preparation*

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Content

Abbreviations

AD	Atypical Depression
ADDS	Atypical Depression Diagnostic Scale
ANOVA	Analysis of Variance
APA	American Psychiatric Association
BABIS	Basel Borderline Inpatient Study
BDI	Beck Depression Inventory
BIS	Barrat's Impulsiveness Scale
BPAQ	Buss Perry Aggression Inventory
BPD	Borderline Personality Disorder
BPI	Borderline Personality Inventory
BRIDGE	Bipolar Disorders: Improving, Diagnosis, Guidance and Education
CLPS	Collaborative Longitudinal Personality Disorders Study
DBT	Dialectical Behavioral Therapy
DSM-III/-IV/-V	Diagnostic and Statistical Manual of Mental Disorders
DST	Disorder Specific Treatment
EKBB	Ethikkommission beider Basel
EOM-EIS	Extended Objective Measure of Ego-Identity Status
GMC	Grey matter concentration
GSI	Global Severity Index
IIP-C	Inventory of interpersonal problems
IPO	Inventory of Personality Organisation
MAOI	Monoamine Oxidase Inhibitors
MBT	Mentalization-based Treatment
NESARC	National Epidemiologic Survey on Alcohol and Related Conditions
PD	Personality Disorder
SCID I/II	Structured Clinical Interview for DSM-IV Axis I and II Disorders
SCL-90-R	Symptom Checklist-90-R
SFT	Schema-focused Therapy
SNF	Swiss National Science Foundation
SPSS	Statistical Package for the Social Sciences
STAI	Spielberger State and Trait Anxiety Inventory
STAXI	Spielberger State and Trait Anger Inventory

STIPO-D	Structured Interview for Personality Organisation
T1-4	Measurement time points
TAU	Treatment as Usual
TEMPS-M	Temperament Auto questionnaire, brief version
TFP	Transference-Focused-Psychotherapy
UPK	Universitäre Psychiatrische Kliniken Basel

Abstract

Borderline Personality (BPD) is a severe psychiatric illness that is characterized by instability in affect regulation, interpersonal relationships, impulse control as well as by a distorted self image. The Basel Borderline Inpatient Study (BABIS) aimed to find out more about this disorder, perhaps even being able to define certain subgroups. On the other hand, a disorder-specific treatment in a specialized unit of the "Universitäre Psychiatrische Kliniken" (UPK) Basel was to be compared to a treatment as usual in other wards of the UPK. All of the following articles emerged from the BABIS-Study.

The first article provides an overview of a specialized form of depression, the so called atypical depression (AD), which often occurs in BPD and shows similarities to it. Included in the article is the German translation of the "Atypical Depression Diagnostic Scale" (ADDS) by Stewart and Colleagues, a special interview to assess AD. The interview was used in addition to the SCID I interview.

In the second article we investigate the relationship of BPD and comorbid AD and found this co-occurrence in nearly a third of the patients. Compared to patients without a comorbid AD, the BPD group with AD shows significant higher scores in anxiety, global severity as well as interpersonal problems. However, there are no differences regarding aggression or impulsivity.

The third article compares borderline patients with high versus low identity diffusion with respect to psychopathology and other Axis II disorders. The results show that patients don't differ in social data but the group with high identity diffusion has significant higher scores in depression, anxiety, anger and general psychiatric symptoms. The same group is also diagnosed more frequently with an additional Axis II disorder, predominantly a Cluster C disorder.

The fourth article explores the outcome of a disorder-specific inpatient treatment (DST) for borderline patients with regard to identity diffusion and psychopathology. This specialized treatment combines a psychodynamic transference-focused psychotherapy (TFP) approach with modules of dialectical behavioural skills training. Compared with treatment as usual (TAU) patients, the DST group shows a significant decrease in identity diffusion as well as anger and depression. However, there are no significant changes for the TAU group.

The role of interpersonal problems in BPD is eventually the subject of the fifth article. Patients with higher interpersonal problems have significant higher scores in identity diffusion and several psychopathological symptoms such as depression or anxiety but not in anger or impulsivity scores. After treatment, all patients show a significant decrease in interpersonal problems as well as in identity diffusion, depression and anger.

In summary the clinical picture of BPD remains complex and we didn't succeed in finding clearly definable subgroups. However it seems that borderline patients do benefit from a disorder-specific treatment. In order to confirm our results, further research with larger study samples would be preferable.

Chapter 1 - Borderline Personality Disorder (BPD)

Borderline personality disorder (BPD) is a serious psychiatric disorder which affects about 0.5% to 5.9% of the general population (Torgersen et al., 2001; Lenzenweger et al. 2007; Trull et al., 2010). It is the most common personality disorder in clinical practice (Skodol et al., 2002a), around 10% outpatients and 15 -25% inpatients are affected (Gunderson, 2009; Torgersen, 2005; Lieb et al., 2004) and about 50% of psychiatric inpatients with personality disorders have a BPD (Lenzenweger, 2008; Lenzenweger et al. ,2007).
Until recently it was assumed that the disorder was more common in women than in men (APA, 2000), however, in the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) with over 34'000 participants (Tomko et al., 2014; Grant et al., 2008) the deviation was found to be only slight. It seems that the gender difference of BPD is primarily caused by the individual characteristics the patients show as well as by comorbid Axis I and II disorders (Sansone & Sansone, 2011; Zlotnick et al., 2002).
Patients with BPD suffer from various symptoms concerning disturbances in identity, affectivity, interpersonal behaviour and impulsivity. (Gunderson & Links, 2008; Sanislow, Grilo, & McGlashan, 2000). Affects of anger and hostility, self-destructive behaviour, elevated stress and negative mood states are characteristic (Henry et al., 2001; Zanarini et al., 1998a). Unfortunately, the risk of suicidality is rather high; at least three-quarters attempt and approximately 10% eventually commit suicide (Black et al., 2004); this rate is 50-times higher than in the general population. BPD patients also show low self-esteem and a distorted self-image (Dammann et al., 2011; Walter et al., 2008).

1.1. Diagnosis

The term „borderline" was expressed for the first time in 1938 by Adolf Stern (1938). It described a group of patients being on the borderline between neurosis and psychosis. Kernberg later developed the term Borderline-Organization (1967) and in 1980, BPD was included for the first time in the DSM-III classification (Fiedler, 2001) containing eight criteria. The classification based on the work of Gunderson and Colleagues (Gunderson & Singer, 1975; Gunderson & Kolb, 1978) and Spitzer, Endicott & Gibbon (1979). Gunderson and Kolb (1978) had developed a diagnostic method to distinguish BPD patients from patients with schizophrenia or depression identifying seven

characteristics; Spitzer and colleagues (1979) then added the eighth criterion "identity disturbance". The ninth criterion was included first in the DSM-IV (APA, 1994).

Current diagnostic criteria

In the DSM-V (APA, 2013), the current diagnostic criteria for BPD is listed as follows:
A pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity beginning by early adulthood and present in a variety of contexts, as indicated by **five (or more) of the following**:

1. Frantic efforts to avoid real or imagined abandonment. Note: Do not include suicidal or self-mutilating behaviour, as it is covered in Criterion 5.
2. A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation.
3. Identity disturbance: markedly and persistently unstable self image or sense of self.
4. Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating). Note: Do not include suicidal or self-mutilating behaviour, which is covered in Criterion 5.
5. Recurrent suicidal behaviour, gestures, or threats, or self-mutilating behaviour.
6. Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days).
7. Chronic feelings of emptiness.
8. Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights).
9. Transient, stress-related paranoid ideation or severe dissociative symptoms.

Additionally, the DSM-V includes another model for further study that takes into account dimensions of self-functioning and interpersonal functioning, particularly impairments in identity or in self-directedness (APA, 2013).

1.2. Comorbidities

Comorbidities are very common in borderline patients, merely a minority show only borderline symptoms. Most of the patients even meet more than one other diagnosis and there is a possibility that these comorbidities can hide an underlying borderline

disorder making it likely for the clinician to overlook the BPD diagnosis entirely which certainly influences the treatment of this patient.

The most common comorbid Axis I disorders are affective as well as anxiety disorders (Tomko et al., 2014; Zanarini et al., 1998a); lifetime prevalence rates for both disorders are over 80% (Tomko et al. 2014). There is also a substantial comorbidity with substance use (Walter et al., 2009) and eating disorders (Zanarini et al., 1998a). More than a third of all patient experience a post-traumatic stress disorder once in their life, hence this comorbidity is rather common but not universal. As mentioned above there seems to be a gender difference regarding comorbid Axis I disorder: female BPD patients are more often diagnosed with a comorbid eating disorder while men are more likely to present with comorbid substance use disorders (Johnson et al., 2003; Tadic et al., 2009).

In the majority of cases, patients with BPD also meet the criteria for at least another Axis II disorder at some point in their lives. In the study of Grant and colleagues (2008), the prevalence rate for personality disorders (PD) in BPD patients was at 73.9 percent, in another study (Barrachina et al., 2011) over 40% of BPD patients had even more than one comorbid Axis II disorder. The most common PDs are Cluster C (mainly avoidant and dependent), paranoid and passive-aggressive PD (Zanarini et al., 1998b). There are also gender differences: women are more likely to be diagnosed with a comorbid dependent PD whereas men show a higher rates of comorbidity with antisocial PD (Barrachina et al., 2011; Zanarini et al., 1998b).

It emerged that these comorbidities might influence each other with regard to the course of the disorder: the Collaborative Longitudinal Personality Disorders Study (CLPS) showed that BPD had a negative effect on the course of substance use disorders (Walter et al., 2009) and affective disorders (Gunderson et al., 2008; Grilo et al., 2010), but these disorders had no, or relatively little, reciprocal effect on the course of BPD. Zanarini and colleagues (2004a) found that borderline patients whose BPD remitted over time also showed substantial decline in comorbid Axis I disorders but for patients without remission the comorbid Axis I disorders remained stable. For Axis II disorders there was a declining rate in both remitted and non-remitted BPD patients for most of the disorders except for Cluster C disorder which remained rather high in the non-remitted BPD group (Zanarini et al., 2004b). Interestingly, the absence of substance use disorders was a strong predictor of remission from BPD (Zanarini et al., 2004a). Several studies (Grilo et al., 2010; Zanarini et al., 2014) found a surprisingly high remission rate

in general for BPD patients: after two years, 60% of the patients still fulfilled the DSM-IV criteria and 33% after 6 years. Zanarini and colleagues (2014) discovered six significant predictors of shorter time to recovery: "no prior psychiatric hospitalizations, higher IQ, good full-time vocational record in 2 years prior to index admissions, absence of an anxious cluster personality disorder, high extraversion, and high agreeableness" (pp. 205).

1.3. Etiology

The cause of BPD is complex and there are several factors that interact with each other (Skodol et al., 2002b), as Lieb et al. (2004) mention: "Genetic factors and adverse childhood experiences might cause emotional dysregulation and impulsivity leading to dysfunctional behaviours and psychosocial conflicts and deficits, which again might reinforce emotional dysregulation and impulsivity" (pp. 454). Thus a biopsychosocial model of personality disorder is assumed (see Figure 1).

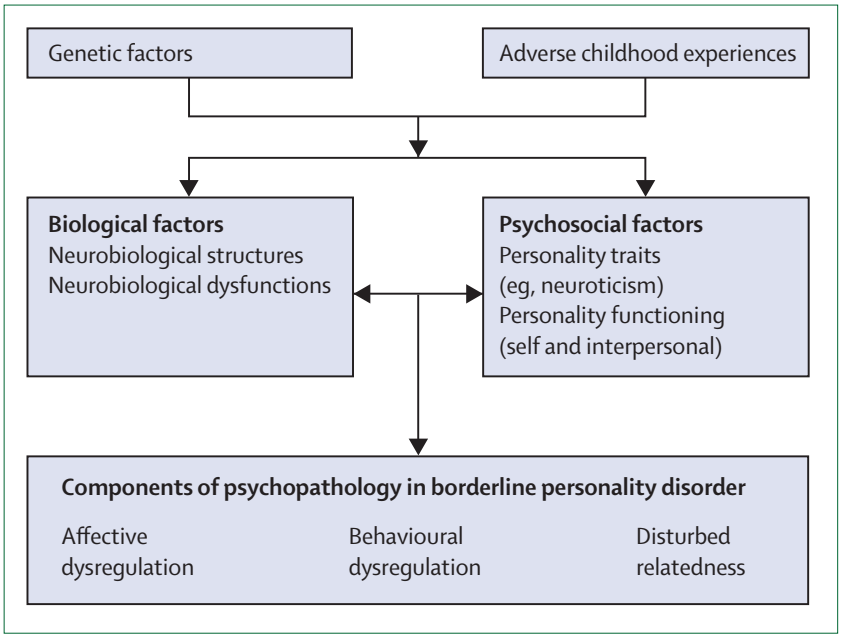

Figure 1: The biopsychosocial model of personality disorder (Leichsenring et al., 2011)

A couple of studies support the influence of genetic factors, the heritability is estimated between 44-69% (i.a. Torgersen et al., 2000). Newer studies are trying to find specific genes that can be linked to BPD[1].

Several neurobiological factors seem to be associated with BPD[2]. A number of studies detected a significant reduction of the hippocampal volume in BPD individuals

1 see Congdon and Canli (2008)

compared to healthy subjects (i.e. Brambilla et al., 2004). Donegan and colleagues (2003) found heightened reactivity of the left amygdala for BPD patients when exposed to facial expression of emotions. In the study of Minzenberg et al. (2008) BPD patients, compared to normal control subjects, had a significant higher grey matter concentration (GMC) in the amygdala as well as a lower GMC in the anterior cingulate cortex. There is also evidence for a hypothalamic-pituitary-adrenal axis dysregulation (Walter et al., 2008; Wingenfeld et al., 2010).

Adverse childhood experiences include abuse (sexual and physical) as well as neglect. In the NESARC-study (Afifi et al., 2011), over 40% of BPD patients reported physical abuse, physical neglect was found in 45% and more than 50% of the patients mentioned general household dysfunction (e.g. battered mother, parent substance use problem). Zelkowitz et al. (2001) found that children who experienced sexual abuse were four times more likely to develop BPD than those who did not.

1.4. Treatment

For some time, the evidence of pharmacotherapy for BPD was rather poor (Zanarini, 2004) and psychotherapy was the principal treatment choice for BPD (APA, 2001). But in the recent past more randomized controlled trials for pharmacotherapy with borderline patients have been conducted[3]. Still the evidence quite varies. Some beneficial effects were reported on depression and aggression but other studies didn't find similar effects, hence more evidence is needed.

There are currently four different forms of psychotherapy that have found to be effective for the treatment of BPD patients: Mentalization-based Treatment (MBT), Transference-focused Psychotherapy (TFP), Dialectical Behavioral Therapy (DBT) and Schema-focused Therapy (SFT). Hereafter these four forms are briefly described otherwise referring to the review articles of Zanarini (2009) and Sollberger & Walter (2010).

Mentalization-based Treatment (MBT)

MBT was developed by Anthony Bateman and Peter Fonagy (2010). They theorize that people with BPD have a decreased capacity for mentalization. Mentalization refers to the capacity to identify the behaviour and feelings of oneself as well as of others. In MBT

2 see O'Neill and Frodl (2012)

3 see Leichsenring et al. (2011)

therapy, the goal is to emphasize the concept of mentalization and to help the BPD patient to increase his mentalization capacity.

Transference-focused Psychotherapy (TFP)

TFP (Clarkin, Yeomans & Kernberg, 1999; 2006) is based on Kernberg's object relations theory. BPD is considered to consist of fragmented representations of self and others that are associated with strong affective experiences which are influenced both by temperament and by difficult early attachment relationships. These fragmented self and object representations lead to fluctuating affects, problematic defence mechanism and disturbed identity and relationships. The primary goal of TFP is to modify these contradictory into integrated representations of self and others.

Dialectical Behavioral Therapy (DBT)

DBT was created by Marsha Linehan (1993) basing on her view that emotional dysregulation was the core feature of BPD. BPD patients are easily upset, run high rapidly and need a fair amount of time to calm down again. This dysregulation leads not only to interpersonal difficulties but also to self-harm or substance abuse. DBT seeks to help patients regulate their emotions, learning about the triggers and provide them with coping skills.

Schema-focused Therapy (SFT)

SFT is based on the work of Jeffrey Young (2003). A schema is an organized pattern of thought and behavior. BPD patients, due to early childhood experiences, develop dysfunctional life schemas that maintain their psychopathology. The goal of SFT is to heal these schemata and replace maladaptive coping styles.

Chapter 2 - The BABIS – Project

2.1. General

The Basel Borderline Inpatient Study (BABIS) was supported by a research grant from the Swiss National Science Foundation (SNF)[4] and took place from 2006 to 2010 in the "Universitäre Psychiatrische Kliniken" (UPK) Basel.
The study was designed as a prospective, non-randomized, two-group comparison inpatient study for patients with a main diagnosis of BPD. The aims of this study were on one side to identify possible subgroups within the heterogeneous group of BPD patients and on the other side to compare the effects of a 12-week disorder-specific treatment (DST) with treatment as usual (TAU).
Written informed consent was obtained from each patient following a full explanation of the study. The study was approved by the local ethics committee (EKBB).
Patients had to be aged between 18 and 65 and diagnosed with a borderline personality disorder according to the DSM-IV-TR criteria. Exclusion criteria were schizophrenia, schizoaffective disorder, active psychosis or an acute manic episode.

2.2. Settings and Treatment

Patients were admitted to the UPK and assigned to the different wards of the clinic[5]. According to this allocation patients willing to take part in our study were then appointed either to the DST-group or the TAU-group.
DST takes place in a specialized psychotherapeutic ward of the UPK and combines a psychodynamic TFP-approach with modules of DBT-training[6]. It combines twice-weekly individual TFP sessions with a primary therapist trained in TFP, together with twice-weekly TFP-oriented psychodynamic group therapy with nurses and a social worker (similar to TAU sessions), as well as weekly supervision and consultation meetings for the therapists. In addition, patients attend weekly DBT-based skills-training groups conducted by trained staff nurses to augment the TFP treatment. DST focuses on both

4 Psychotherapy Outcome and Subgroups in Disorder- Specific Inpatient Treatment of Borderline Personality Disorder: A Prospective Controlled Matched-Sample Study (32003B-108462)

5 without respect to the study

6 see section 1.4. Treatment

the decline in psychopathological symptoms and on an improvement in structural features of personality organization, particularly changes in identity diffusion[7].
TAU consists of clinical management (supportive treatment, social psychiatry and psychopharmacotherapy) by the psychiatric services. Patients in this group generally attend one non-specific psychotherapeutic session per week with a psychiatrist, psychoeducation in group therapy, supportive talks with staff nurses and one session per week with a social worker. Once a week, the senior physician of the unit supervises the staff team. All team members are experienced in treating patients with BPDs but not trained in specialized evidence-based treatments of this disorder.
DST has a fixed duration of stay of 12 weeks whereas the duration in the TAU wards is variable. In our study, the mean duration of stay for the TAU patients was at 11.33 weeks. Thus, treatment dose[8] expressed as period of treatment was comparable in both groups but slightly in favour of DST.

2.3. Schedule and Research Plan

When entering the clinic, potential patients for both groups were asked to participate in the study (see Figure 2). If they agreed a first interview to determine Axis I and II disorders (SCID I/II; ADDS[9]) was conducted during the first week of their stay and additionally, patients had to fill out the questionnaire[9]. Another interview concerning personality organisation (STIPO-D[9]) took place a few days later and thus T1 was completed.
The STIPO interview was recorded on video in order to measure facial affective behaviour of both the patient and the interviewer (Split-Screen-Technique). This part of the study was in cooperation with the University of Innsbruck (Prof. Dr. Benecke and colleagues) and they conducted the analysis of the recordings.
At the end of treatment (usually after 12 weeks), for T2, the STIPO interview was conducted one more time (also with recording) and the patients had to complete the same questionnaire as before. T3 took place 6 month after discharge and contained only the questionnaire. Another 6 month later, thus a year after treatment, patients were asked to fill-out the questionnaire once more for T4.

7 for more see Sollberger et al. (2014)
8 Howard, Kopta, Krause, & Orlinsky (1986)
9 More detailed information see section „Clinical Measurements“ below

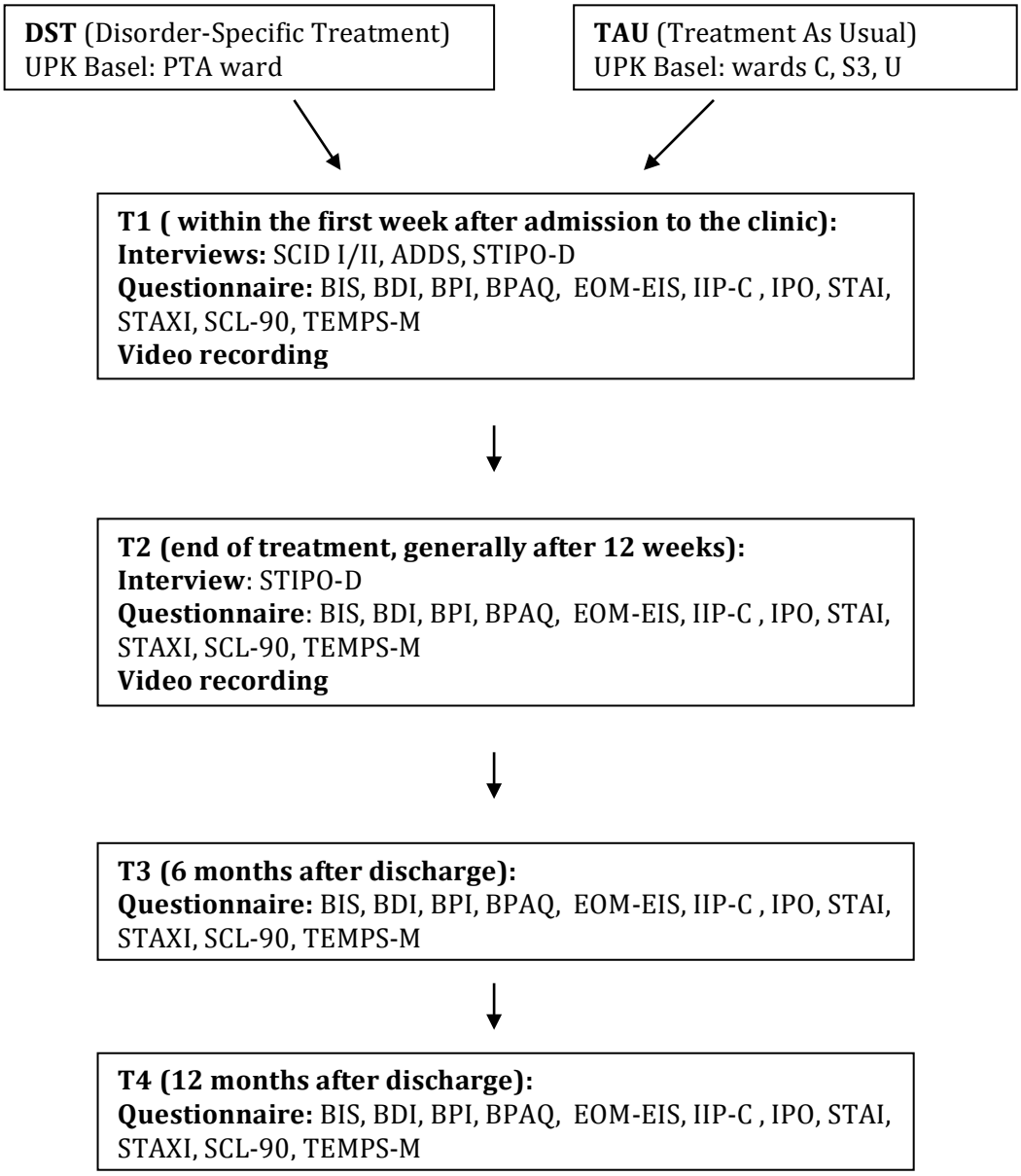

Figure 2: Schedule and research plan of the BABIS - Project

2.4. Clinical measurements

Interviews

Clinically experienced interviewers were trained for structured clinical interviewing. To identify Axis I and Axis II disorders and confirming an actual BPD diagnosis, the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P) (First et al., 1996) and for DSM-IV Axis II Disorders (SCID-II) (First et al., 1997) was used. Both semi-structured interviews show high interrater reliability (Lobbestael, Leurgans & Arntz, 2011; Maffei et al., 1997).

Additionally, the Atypical Depression Diagnostic Scale (ADDS) (Stewart et al., 1993) was used to examine atypical depression in a more detailed way. The ADDS is a semistructured interview designed to investigate the presence and severity of atypical features during current depressive episodes. The ADDS was translated into German especially for this study (Gremaud-Heitz et al., 2011).
Furthermore the Structured Interview for Personality Organization (STIPO) (Clarkin et al. 2003; Doering, 2013) was conducted. The STIPO is an semi-structured instrument to assess personality organization basing on Kernberg's psychodynamic concept (1984) postulating three levels of personality organisation: neurotic, borderline, and psychotic. On a dimensional level identity, object relations, primitive defences, coping/rigidity, aggression, moral values and reality testing are determined. In our study, only the identity part of the interview was performed.

Questionnaire

Each patient completed a questionnaire with the following instruments:

- **Barratt's Impulsiveness Scale (BIS)** (Barratt, 1965; Meule et al., 2011)
- **Beck Depression Inventory (BDI)** (Beck et al., 1961; Hautzinger et al., 2000; Dozois et al., 1998)
- **Borderline Personality Inventory (BPI)** (Leichsenring, 1997; Leichsenring & Chabrol, 2006)
- **Buss Perry Aggression Inventory (BPAQ)** (Buss & Durkee 1957; Buss & Perry 1992)
- **Extended Objective Measure of Ego-Identity Status (EOM-EIS)** (Adams, 1989; Schwartz, 2004)
- **Inventory of Interpersonal Problems (IIP-C)** (Horowitz et al., 1988; Horowitz et al., 2000)
- **Inventory of Personality Organisation (IPO)** (Clarkin et al.,2000; Dammann et al., 2002; Lenzenweger et al.,2001)
- **Spielberger State and Trait Anxiety Inventory (STAI)** (Spielberger et al., 1970; Laux et al., 1981; Barnes et al., 2002)
- **Spielberger State and Trait Anger Inventory (STAXI)** (Spielberger, 1988; Schwenkmezger et al., 1992; Mueller et al., 2001)
- **Symptom Checklist-90-R (SCL-90-R)** (Derogatis, 1983; Franke, 1995)

- **Temperament Auto questionnaire, brief version (TEMPS-M)** (Erfurth et al., 2005; Akiskal et al., 2005)

All these psychometric instruments are used widely with generally good reliability and validity[10].

2.5. Hypotheses

The original proposal of the study included several hypotheses[11] divided into the three fields: 1., subgroups, 2., psychotherapy, and 3., treatment response. As mentioned above, the part of the project concerning facial expression was in cooperation with the University of Innsbruck hence the following hypotheses on this subject were evaluated by Benecke and colleagues[12].

Subgroups

- BPD patients can be divided into those with severe identity disorder and fewer expressed emotions and those with less identity disturbances, more expressed emotions and severe affective disturbances
- BPD patients who overwhelmingly exhibit facial expressions of happiness show significantly higher identity diffusion than BPD patients with "aggressive" affective facial expression
- BPD patients fulfilling criterion 6 of the DSM-IV-BPD-Criteria (affective instability due to a marked reactivity of mood) show significantly more characteristics of atypical depression and/or cyclothymic-bipolar-spectrum

Psychotherapy

- Compared to the TAU-group, the 12-week DST leads to a significant reduction of depressivity, impulsivity, symptom complaints and negative affects
- Improvement at the end of the therapy will be maintained six month after treatment in the DST but not in the TAU group
- BPD patients of the DST group will have less hospitalisation days than the TAU group in the first year after treatment

10 information on reliability and validity of the instruments can be found in the quoted literature

11 pp. 9-10

12 article in preparation

- Only BPD patients of the DST group will fulfil significantly less severity criteria for BPD six month after treatment compared to the beginning

Therapy response

- BPD patients who exhibit a higher extent of identity diffusion at the beginning show worse therapy outcomes than those with lower extent in both treatment groups
- BPD patients who exhibit more negative affective facial expression at the beginning show a better therapy outcome than those with fewer expressions in both treatment groups
- The BPD group who exhibits more severe depression and/or impulsivity show no worse therapy outcome than the patients with less severe depression and/or impulsivity

2.6. Statistical Analyses

All statistical analyses were conducted with the respective current version of the computer software „Statistical Package for the Social Sciences“ (SPSS). Assumption of homoscedasticity and normality distribution were checked prior to the analysis. χ^2-Tests were used for the testing of categorical data. Comparison of two groups was conducted with Student's t-Test for normally distributed data and Mann Whitney U-Test if a normal distribution wasn't given. Univariate ANOVA was performed to compare more than two independent groups. For group comparison before and after treatment, we ran a paired t-Test (normal distributed data) and Wilcoxon signed rank (no normal distribution assumed) respectively. All tests results were considered significant at a two-sided level of $p \leq 0.05$.

2.7. Patients

70 Patients were invited to participate in the study. Due to different reasons only 60 patients performed the first part of our study (T1) (see Figure 3). Unfortunately, another 11 patients dropped out of the study at the end of treatment (T2). 6 months after treatment it became far more difficult to contact the remaining patients after they had left the clinic and the willingness to further participate decreased considerably; another 18 patients didn't complete T3. Not surprisingly there were 11 drop-outs for T4. Only 15

out of 60 Patients completed all four assessment points. Therefore, it became clear that the statistical calculation would only be reasonable for T1 and T2, hence some of the original hypotheses mentioned above became obsolete.

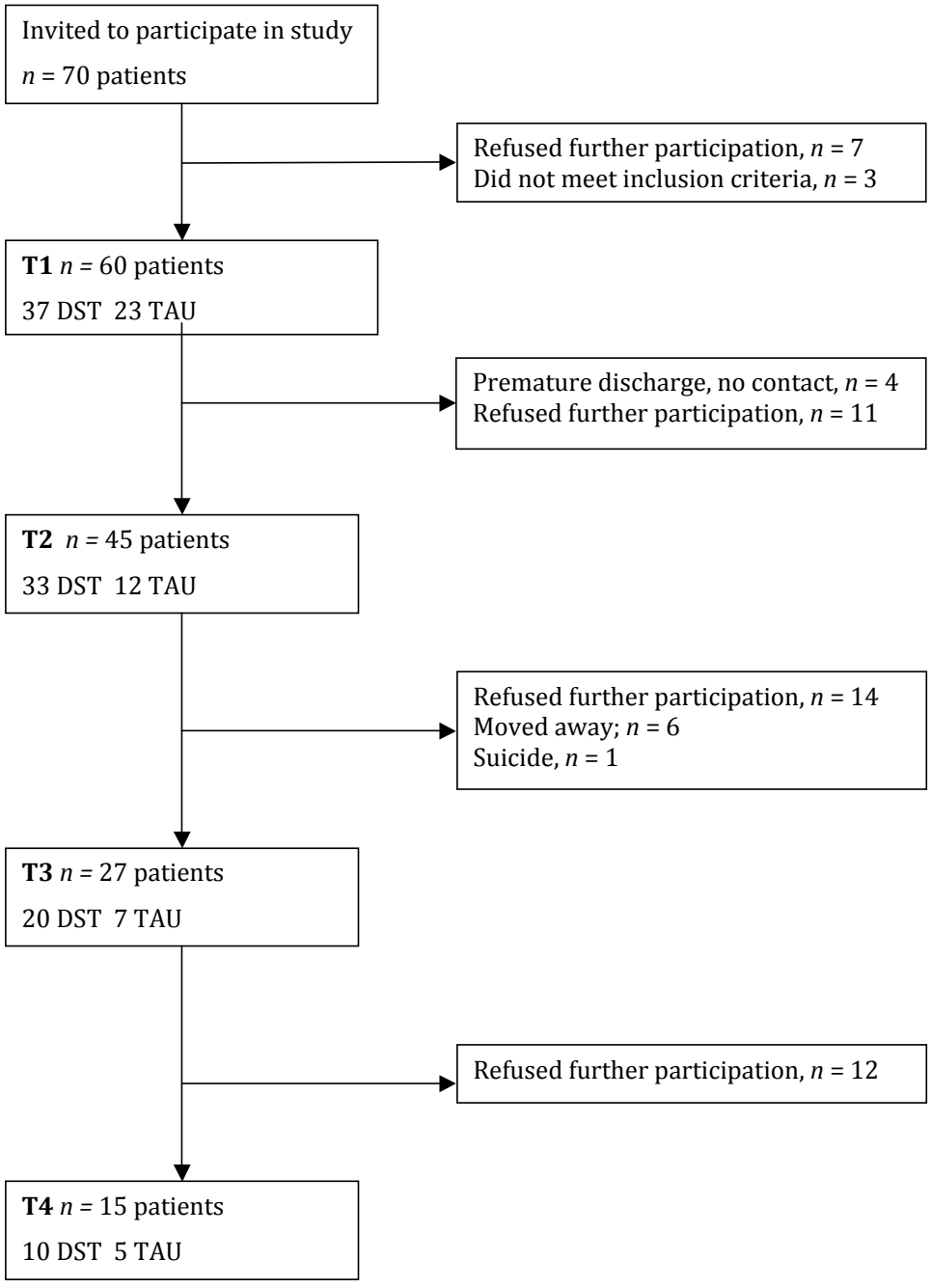

Figure 3: Number of respondents and response rates at baseline and follow-ups

Demographic and Clinical Data

Of the 60 patients that entered our study, 49 were women and 11 men. The youngest patient was 18 years, the oldest 53 years, the mean age was 28 years (see Table 1). Most of the patients were single and only 5 patients had children.

Table 1 :
Demographic characteristics of all patients and for both groups separately

	Patients (n=60)		
		DST (n=37)	*TAU (n=23)*
Age, mean (SD)	28.95 (8.7)	26.66 (6.3)	32.65 (10.7)
Gender, *n (%)*			
Female	49 (81.7)	30 (81.1)	19 (82.6)
Male	11 (18.3)	7 (18.9)	4 (17.4)
Marital status, *n (%)*			
Single	37 (61.7)	26 (70.3)	11 (47.8)
Relationship/Married	11 (18.3)	5 (13.5)	6 (26.1)
Separated/Divorced	7 (11.7)	3 (8.1)	4 (17.4)
n/a	5 (8.3)	3 (8.1)	2 (8.7)
Children, *n (%)*			
None	44 (73.4)	27 (73)	17 (73.9)
1-2	11 (18.3)	7 (18.9)	4 (17.4)
n/a	5 (8.3)	3 (8.1)	2 (8.7)
Education, *n (%)*			
None	1 (1.7)	-	1 (4.3)
< 9 years	22 (36.7)	12 (32.4)	10 (43.5)
9 - 12 years	20 (33.3)	16 (43.3)	4 (17.4)
>12 years	11 (18.3)	6 (16.2)	5 (21.7)
n/a	6 (10)	3 (8.1)	3 (13.1)
Employment, *n (%)*			
Employed (full/part time)	12 (20)	11 (29.8)	1 (4.3)
Apprenticeship	9 (15)	8 (21.6)	1 (4.3)
Unemployed	15 (25)	9 (24.3)	6 (26.1)
Disability pension	12 (20)	5 (13.5)	7 (30.5)
Pension & employed	6 (10)	1 (2.7)	5 (21.7)
n/a	6 (10)	3 (8.1)	3 (13.1)

Note. n/a= not applicable ; SD= Standard Deviation

The differences of the treatment groups in demographical features were statistically not significant except for current employment (p=0.011). All patients were on medication deemed appropriate by the psychiatrists and in accordance with the recommended APA guidelines (Soloff, 2000).

As can be seen in Table 2, comorbid disorders were very common in our patients, only two DST patients weren't diagnosed with a comorbid Axis I disorder. Most frequent in both groups were affective disorders (83.3.%), followed by substance related disorders (63.3%). Almost 65% of the patients had a comorbid Axis II disorder diagnosis, most

commonly a Cluster C disorder. Over 25% of the patients were diagnosed with 3 or more comorbid Axis II disorders.

Table 2
Clinical Characteristics of all patients and for both groups separately

	Patients (n=60)		
		DST (n=37)	*TAU (n=23)*
Comorbid Axis I Disorder, *n (%)*			
None	2 (3.3)	2 (5.4)	-
Affective disorder	50 (83.3)	31 (83.8)	19 (82.6)
Anxiety disorder	32 (53.3)	23 (62.2)	9 (39.1)
Substance related disorder	38 (63.3)	23 (62.2)	15 (65.2)
Eating disorder	22 (36.6)	15 (40.5)	7 (30.4)
Comorbid Axis II Disorder, *n (%)*			
Quantity			
None	21 (35)	11 (29.7)	10 (43.5)
1-2	21 (35)	14 (37.9)	7 (30.4)
3-4	16 (26.7)	12 (32.4)	4 (17.4)
n/a	2 (3.3)	-	2 (8.7)
Comorbid Axis II Disorder, *n (%)*			
Form			
Cluster A	11 (18.3)	6 (16.2)	5 (21.7)
Cluster B	6 (10)	2 (5.4)	4 (17.4)
Cluster C	30 (50)	24 (64.9)	6 (26.1)
Passiv-Aggressive	8 (13.3)	7 (18.9)	1 (4.3)
Depressive	16 (26.7)	12 (32.4)	4 (17.4)
None	21 (35)	11 (29.7)	10 (43.5)
n/a	2 (3.3)	-	2 (8.7)
Duration of illness, *n (%)*			
<1 year	4 (6.7)	2 (5.4)	2 (8.7)
1 year to 5 years	17 (28.3)	13 (35.1)	4 (17.4)
5 to 10 years	8 (13.3)	5 (13.5)	3 (13)
10 to 20 years	18 (30)	10 (27.1)	8 (34.9)
>20 years	7 (11.7)	4 (10.8)	3 (13)
n/a	6 (10)	3 (8.1)	3 (13)
Previous therapies, *n (%)*			
None	11 (18.3)	6 (16.2)	5 (21.7)
Ambulant treatment	6 (10)	5 (13.5)	1 (4.3)
Inpatient treatment	9 (15)	5 (13.5)	4 (17.4)
Ambulant + inpatient treatment	29 (48.4)	18 (48.7)	11 (47.9)
n/a	5 (8.3)	3 (8.1)	2 (8.7)

Notes. n/a= not applicable

Only in a minority of the patients (n=4) the duration of illness was under a year, for over 40% it was 10 years and more. Approximately half of the patients already has had ambulatory and stationary treatment, only for 11 patients it was the first treatment. Comparison of both treatment groups only showed a significant difference in comorbid Cluster C disorder (p= 0.006).

Chapter 3 - Results of the BABIS - Project

As mentioned before, BPD is a very heterogeneous disorder. Since there are nine different criteria and only five of them need to be fulfilled for a diagnosis there are at least 126 different possibilities (clusters) to meet the diagnostic criteria for BPD (Korfine & Hooley, 2009). The nine criteria can be organized into four sectors of psychopathology: affective, cognitive, behavioural and interpersonal criteria (Lieb et al., 04). Patients vary widely in their severity of manifestation of these factors and even do not need to be impaired in all four sectors.
Below I will go into detail on the five articles that have resulted from the project and to which I have contributed substantially. All five articles are listed in the declaration as well as in the appendix and will now be discussed in turn.

3.1. Affectivity (Articles 1[13] and 2[14])

Background

Affect dysregulation is one of the core features of BPD. It refers to the inability of a person to control or regulate his or her emotional responses to provocative stimuli. As a consequence of this, emotions spiral out of control, change rapidly and get expressed in intense and unmodified forms (Conklin, Bradley & Westen, 2006). Linehan (1993), proposes that this vulnerability to emotion dysregulation in BPD is characterized by high sensitivity to emotional stimuli, high emotional intensity, and slow return to emotional baseline once emotional arousal has occurred.
Yen, Zlotnick and Costello (2002) were able to show that BPD patients experienced emotions more intensely and had greater difficulty in controlling their affective responses. Conklin and colleagues (2006) compared BPD patients with patients with dysthymic disorder and found that both showed negative affects whereas only the BPD group was characterized by affect dysregulation. In a different study Trull et al. (2008) explored affective instability in patients with BPD and patients with a depression. They found that both groups showed relatively high levels of negative affect. However, the groups did differ significantly in the degree of variability of the level of negative affect with BPD patients having higher frequencies in mood change.

13 Appendix 1 (Gremaud-Heitz, Stewart & Dammann, 2011)
14 Appendix 2 (Gremaud-Heitz et al, 2014)

According to Perugi and colleagues (2011) this affective dysregulation is also the basis for a specific subgroup of depression, the atypical depression (AD), which can influence a possible diagnosis: "the 'atypicality' of depression is related to an affective temperamental dysregulation, which could explain why atypical depressive patients are often given 'borderline' diagnoses" (pp. 45).

In the study of Posternak & Zimmermann (2002) almost a third of the BPD patients were diagnosed with a comorbid AD. The more recent international BRIDGE-study (Perugi et al., 2013) found more atypical features in depressed patients with a comorbid BPD whereas a diagnosis of a bipolar disorder was more common in the group without BPD.

AD was introduced to specify major depressive episodes in DSM-IV following a series of antidepressant trials showing that such patients responded preferentially to monoamine oxidase inhibitors (MAOI's) (Paykel, 1993). This depression form is characterized by depressive mood, emotional reactivity, increased sleep, eating disorders as well as somatic impairment. AD affects about 30% of unipolar depressive patients, mostly women, and, compared to other depression forms, shows an earlier age of onset and a more chronic course of illness (Stewart et al., 2007). Further information on AD can be found in Article 1[15].

The symptom of heightened rejection sensitivity (Staebler et al., 2011)is characteristical for both BPD and AD. Anxiety seems to be an important factor for both disorders too. There are high comorbidity rates for both BPD (Silverman et al., 2012; Comtois et al., 1999) and AD (Gili et al., 2012; Novick et al., 2005).

Article 2[16] investigated the co-occurrence of AD in BPD-patients and compared this group to BPD patients with a different comorbid depression or patients with no depression.

Results

Forty-five patients (81.8%) had a comorbid affective disorder of which 15 (27.3%) were diagnosed with an atypical depression. All of them fulfilled the BPD criterion 6[17]. In comparison to patients with other depression or no depression at all, AD patients showed significantly higher scores in several psychopathological symptoms particularly

15 Appendix 1 (Gremaud-Heitz, Stewart & Dammann, 2011)

16 Appendix 2 (Gremaud-Heitz et al., 2014)

17 affective instability due to a marked reactivity of mood

anxiety and depression (see Table 3). There were also significant differences in interpersonal problems primarily with scales on the submission dimension. However, there were no differences in anger or hostility.

Table 3:
Intergroup Differences regarding Psychopathology[18]

	*Group1 Atypical Depression (*n*=15)*	*Group 2 Other Depression(* n*=30)*	*Group 3 No Depression (*n*=10)*	*F-value (*p*)*
SCL-90, mean *(SD)*				
GSI	1.7 (0.6)	1.3 (0.7)	1.1 (0.6)	4.936 (.011)*
Somatization	16.2 (11.4)	11.0 (7.8)	10.0 (5.8)	2.658 (.080)n.s.
Obsess.-Compulsive	18.7 (8.4)	13.7 (7.6)	10.1 (4.6)	6.910 (.002)**
Interpers. Sensitivity	18.1 (7.1)	14.1 (8.4)	11.3 (6.2)	3.890 (.027)*
Depression	31.0 (10.1)	24.9 (12.4)	18.1 (7.8)	7.092 (.002)**
Anxiety	18.4 (7.5)	12.1 (7.9)	12.5 (7.4)	3.874 (.027)*
Hostility	9.1 (5.5)	7.5 (5.4)	6.6 (5.3)	1.022 (.367)n.s.
Phobic Anxiety	13.2 (7.5)	7.3 (6.9)	5.7 (5.5)	6.269 (.004)**
Paranoid Ideation	8.7 (4.0)	6.6 (5.3)	6.2 (5.6)	1.342 (.270)n.s.
Psychoticism	11.5 (8.3)	9.5 (7.5)	8.5 (7.1)	0.704 (.499)n.s.
BDI, mean *(SD)*				
Depression Score	30.3 (8.5)	25.7(11.5)	17.5 (9.9)	7.151 (.002)**
STAI, mean *(SD)*				
State Anxiety	59.9 (11.1)	51.1 (13.9)	48.5 (12.3)	6.961 (.002)**
Trait Anxiety	62.4 (6.9)	55.2 (11.4)	50.5 (11.8)	8.501 (.001)**
STAXI, mean *(SD)*				
State Anger	20.7 (9.3)	16.0 (5.9)	15.1 (7.1)	2.804 (.070)n.s.
Trait Anger	21.9 (6.9)	21.8 (7.7)	19.4 (5.9)	0.773 (.467)n.s.
IIP, mean *(SD)*				
Total Score	2.0 (0.4)	1.8 (0.6)	1.3 (0.6)	6.548 (.003)**
Domin./Controlling	5.5 (3.8)	7.6 (4.8)	6.1 (4.9)	0.955 (.392)n.s.
Vindic./Self-Centered	11.2 (3.7)	11.5 (5.6)	10.1 (5.5)	0.380 (.686)n.s.
Cold/Distant	12.5(5.1)	13.3 (6.3)	11.3 (5.3)	0.562 (.574)n.s.
Socially Inhibited	20.1(6.5)	16.8 (7.1)	12.4 (5.8)	6.440 (.003)**
Nonassertive	21.5 (8.2)	16.4 (7.1)	9.9 (8.2)	9.924 (.000)**
Ov. Accommodating	20.0 (5.9)	15.2 (7.9)	11.5 (5.2)	8.160 (.001)**
Self-Sacrificing	21.8 (5.4)	18.8 (7.7)	14.3 (5.8)	6.476 (.003)**
Intrusive/Needy	12.5 (4.3)	12.6 (6.6)	9.6 (6.3)	1.537 (.225)n.s.

*Notes. SD= standard deviation, n.s.: non significant, * p<0.05, ** p<0.01*

3.2. Identity (Article 3[19])

Background

Identity disturbance is one of the core features of BPD. In DSM-V (APA, 2013), this criterion is described as a markedly and persistently unstable self-image or sense of self. Most of the BPD patients report a strong uncertainty of their own identity. Approximately 70% report that they "don't know who they really are" (Bohus, 2002).

18 pp.653

19 Appendix 3 (Sollberger et al., 2012)

Erikson (1956) was the first to formulate the term identity diffusion. He described it as an absence or loss of the normal capacity for self definition resulting in isolation, a sense of inner vacuum and regression to earlier identifications. In Kernberg's view (2006), identity diffusion is one of the three components of personality organization (next to primitive psychological defences and reality testing) and thus the level of an individual's personality organization is dependent on the persons position on each of this three parts. He described identity diffusion as: „a structural, pathological consolidation of the internalized world of object relations, reflected in a stable lack of integration of the concept of self and of significant others" (pp.980). Other authors mention lack of long term goals, negative self-image and lack of sense of continuity in self-perception over time (Jorgensen, 2006; Fuchs, 2007).

In contrast to the typical identity crisis of an adolescent, a BPD patient isn't able to describe himself or important people in his life in a realistic way. Internal value systems are absent or the patient shows a chaotic or conflicting attitude towards them. In the qualitative research study of Dammann and colleagues (2011), BPD patients characterized themselves almost exclusively with positive attributes (e.g. friendly, helpful, caring); others were described predominantly with negative features (e.g. selfish, evil). A depressive comparison group on the other hand was able to picture themselves and others in a far more sophisticated way being able to both describe positive and negative qualities.

Wilkinson-Ryan and Westen (2000) were able to show that identity disturbance distinguishes BPD from patients with other PD's or no PD. Compared with depression, BPD patients show higher levels of self-criticism (Levy et al., 2007) and even in comparison to social phobia, BPD report a lower self- esteem (Rüsch et al., 2007). Lenzenweger and colleagues (2001) found that identity diffusion was significantly correlated with negative affects and irritability. And a study with juvenile offenders (Dammann et al., 2011) found a significant correlation between identity diffusion and aggression as well as critical alcohol consumption.

However, until now there hasn't been a study that investigated the influence of the severity of identity diffusion on other factors.

Article 3[20] aimed at comparing patients with high and low identity diffusion with regard to psychopathology. A median split of the IPO subscale „identity diffusion" was conducted to build the two groups.

Results

BPD patients with high identity diffusion did not differ in their social data from BPD patients (see Table 4). However, the group with high identity diffusion had significant more comorbid Cluster C disorders and more comorbid Axis II disorders on average. There were no significant differences in any comorbid Axis I disorder.

Table 4 :
Social and clinical data of patients with high and low identity diffusion[21]

	*High identity diffusion(*n=*27)*	*Low identity diffusion(*n=*25)*	*p*
Age, mean (SD)	28.5.6 (8.0)	29.4 (9.4)	n.s.
Sex, n *(%)*			
Male	6 (22.2)	4 (16.00)	n.s.
Female	21 (77.8)	21 (84.00)	
Job situation, n *(%)*			
Employed	8 (29.6)	5 (20.0)	n.s.
Unemployed	19 (70.4)	20 (80.0)	
Family situation, n *(%)*			
Living with a partner	8 (29.6)	5 (20.0)	n.s.
Living alone	19 (70.4)	20 (80.0)	
Duration of BPD diagnosis, n (%*)*			
<1 year	-	3 (12.0)	n.s.
1-5 years	8 (29.6)	8 (32.0)	
>5 years	19 (70.4)	14 (56.0)	
Axis I disorder, n (%*)*			
None	1 (3.7)	1 (4.0)	n.s.
Affective disorder	24 (88.9)	18 (72.0)	n.s.
Anxiety disorder	15 (55.6)	12 (48.0)	n.s.
Substance use disorder	17 (63.0)	16 (64.0)	n.s.
Eating disorders	11 (40.7)	8 (32.0)	n.s.
Axis II disorder (Form), n (%*)*			
None	4 (14.8)	13 (52.0)	χ^2=8.37, p=0.04
Cluster A	5 (18.5)	4 (16.0)	n.s.
Cluster B	4 (14.8)	1 (4.0)	n.s.
Cluster C	18 (66.7)	8 (32.0)	χ^2=8.37, p=0.04
Axis II disorder (quantity), n (%*)*			
None	4 (14.8)	13 (52.0)	χ^2=8.37, p=0.04*
1-2	12 (44.4)	9 (36.0)	n.s.
3-4	10 (37.1)	2 (8.0)	χ^2=6.17, p=0.013*
n/a	1 (3.7)	1 (4.0)	n.s
Axis II disorder (quantity), mean (SD)	2.00 (1.41)	0.75 (0.99)	Z= -3.26, p=0.001**

*Notes. SD= standard deviation n.s.=non significant, * p<0.05, ** p<0.01*

20 Appendix 3 (Sollberger et al., 2012)

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The comparison of questionnaire data showed that BPD patients with high identity diffusion reported significantly higher levels in all personality organisation scales, as well as higher anxiety, anger, and depression scores, all of them even on a 1% significance level (see Table 5).

Table 5:
Identity diffusion and negative affects[22]

	*High identity diffusion(*n=*27)*	*Low identity diffusion(*n=*25)*	*p*
IPO, mean (*SD)*			
Primitive defences	48.16 (5.76)	35.28 (7.64)	t= -6.90, p<0.0001**
Identity diffusion	68.60 (8.02)	46.44 (10.64)	t= -8.52, p<0.0001**
Reality testing	48.70 (12.46)	32.36 (8.70)	t= -5.44, p<0.0001**
Aggression	37.05 (7.91)	28.25 (4.94)	t= -4.77, p<0.0001**
Moral values	28.45 (7.03)	21.00 (6.05)	t= -4.08, p<0.0001**
SCL-90-R, mean *(SD)*			
Global severity index	1.79 (0.58)	0.98 (0.56)	t= -5.16, p<0.0001**
BDI, mean *(SD)*			
Depression Score	29.26 (9.73)	20.44 (11.29)	t= -3.02, p=0.004**
STAI, mean *(SD)*			
State Anxiety	58.84 (11.55)	48.67 (12.93)	t= -2.99, p=0.004**
Trait Anxiety	61.56 (8.42)	50.38 (11.21)	t= -4.08, p<0.0001**
STAXI, mean *(SD)*			
State Anger	20.45 (8.68)	14.40 (4.3)	t= -3.22, p=0.003**
Trait Anger	24.19 (7.11)	18.04 (5.7)	t= -3.43, p=0.001**

*Notes. SD= standard deviation, * p<0.05, ** p<0.01*

3.3. Treatment Response (Article 4[23])

Background

Several operationalized and evidence-based disorder-specific outpatient treatments have shown effects in symptom reduction and enhancement in general functioning (Sollberger & Walter, 2010; Stoffers et al. 2012). As mentioned before, the disorder-specific treatment that our study patients attended combined TFP with DBT-modules. The efficacy of both these treatments has been shown in several trials (e.g. Doering et al., 2010; Linehan et al., 2006). However, the vast majority of these studies have been

22 pp. 18

23 Appendix 4 (Sollberger et al., 2014)

performed in an outpatient setting. But there is evidence that DBT is also effective in inpatient treatment (Bloom et al., 2012). The benefit of DBT lies particularly in the improvement of self-harm behaviour and the reduction of depressive symptoms and suicidal ideation (Katz et al., 2004). TFP on the other hand shows additional improvement in anger, impulsivity as well as in personality organisation (Clarkin et al., 2007; Doering et al., 2010), but until now, all TFP studies were conducted with outpatients.

Therefore article 4[23] sought to investigate the effects of a DST treatment containing TFP aspects in an inpatient setting. Patients were compared with a TAU group and the primary focus of the study were changes in identity diffusion and psychopathological symptoms.

Results

After 12 weeks, the DST patients showed a significant decrease in identity diffusion (p= 0.006), instability in self/others (p=0.008), depression (p=0.002) and anger (p=0.001). However, there was no significant improvement in the TAU group, they even showed an increase in several variables (identity diffusion, instability, anger) though not significant (see Table 6).

Table 6:
Pre-post-tests of DST- (n=32) and TAU- (n=12) group[24]

Variable	*Mpre*	*SDpre*	*Mpost*	*SDpost*	*t*	*p*
IPO (theoretical construct)						
Primitive defences - DST	43.42	8.94	41.89	10.63	1.24	0.225
Primitive defences - TAU	37.41	9.08	36.92	7.96	0.27	0.789
Identity diffusion - DST	62.71	12.81	58.49	14.01	2.95	0.006**
Identity diffusion - TAU	47.75	13.74	48.92	12.28	-0.38	0.711
Reality testing -DST	41.19	12.67	39.99	13.98	0.43	0.673
Reality testing- TAU	38.83	16.66	37.70	12.21	0.42	0.683
Aggression - DST	34.11	8.21	33.12	7.95	0.98	0.337
Aggression - TAU	32.27	7.12	29.88	7.67	2.15	0.060
Moral values - DST	25.59	7.80	24.67	6.98	0.93	0.358
Moral values - TAU	21.83	5.94	20.83	5.88	0.73	0.481
IPO (four-factor structure)+						
Instability in self/others - DST	94.41	17.95	88.35	21.61	2.85	0.008**
Instability in self/others - TAU	75.33	19.90	77.35	17.80	0.46	0.964
Instability in goals - DST	5.61	2.38	5.27	2.62	1.07	0.293
Instability in goals - TAU	4.75	2.65	5.82	2.69	-1.70	0.120
Psychosis -DST	21.42	8.65	21.11	9.28	0.20	0.840
Psychosis- TAU	20.67	9.70	19.54	7.45	1.16	0.274
Instability in behaviour - DST	19.10	5.56	18.99	5.65	-0.22	0.832
Instability in behaviour - TAU	16.40	6.81	15.91	6.24	1.27	0.233
BDI						
Depression Score - DST	27.16	9.16	21.39	12.31	3.65	0.002**
Depression Score - TAU	23.64	12.92	18.86	14.49	0.94	0.370
STAI						
State Anxiety. - DST	56.25	11.00	53.10	12.86	1.36	0.184
State Anxiety - TAU	50.70	14.86	49.33	14.03	0.41	0.690
STAXI						
State Anger - DST	18.56	8.07	14.28	5.26	-3.18	0.001**
State Anger - TAU	15.00	6.83	17.18	8.38	-1.67	0.126

+by Ellison & Levy, 2012
*Notes. M= Mean, SD= standard deviation, * p<0.05, ** p<0.01*

3.4. Interpersonal Problems (Article 5[25])

Background

BPD patients have major problems in interpersonal relations. Relationships are instable basing on the contrasting anxieties of a BPD patient; fear of closeness and fear of loneliness respectively. It is difficult for persons with BPD to regulate closeness and distance which not only leads to problems in friendships, but also makes it difficult at work and of course complicates the therapist-patient relationship.
Although interpersonal problems occur generally in personality disorders, BPD patients were found to report higher interpersonal dysfunction scores (Stepp et al., 2011) than patients with other PD's.

24 pp. 7

25 Appendix 5 (Dammann et al., in preparation)

BPD patients show a wide variety of interpersonal problems (Zanarini & Frankenburg, 2007) in contrast to other PD's, where correlations with specific interpersonal problem patterns can be found (Clarkin et al., 2011). However, Hilsenroth and colleagues (2007) found BPD patients to report greater interpersonal distress in the sectors "overly accommodating", "self-sacrificing" and "intrusive-needy" in comparison to a clinical control group. And Leihener et al. (2003) even described two distinct subtypes in BPD patients; the "autonomous" and the "dependent" type (pp. 251/252). A German study with over 200 BPD inpatients (Salzer et al., 2013) found five different interpersonal clusters and showed that these had a significant influence on interpersonal distress and global severity symptoms. Hence it seems that BPD patients are interpersonally heterogeneous and cannot be characterized with a specific interpersonal style (Wright et al., 2013a).

Interpersonal problems are a central focus of treatment and perist even after other symptoms have remitted (Clarkin, Yeomans & Kernberg, 1999; Zanarini et al., 2007). Wright and colleagues (2013b) investigated the stability of interpersonal problems in BPD patients over the course of a year and found that „interpersonal dysfunction on borderline pathology is stable in its severity but unstable in the style of its manifestation" (pp. 1094).

Thus article 5[26] explored interpersonal problems in borderline patients whether the severity of interpersonal problems had an influence on other borderline features and psychopathology. For this article, we analyzed only the patients of the DST group. To compare the severity of interpersonal problems, we conducted a median split of the IIP general scale. The second part of the article addresses the question whether interpersonal problems diminish during treatment.

Results

Comparison of the two groups showed that patients with higher interpersonal problems reported significant higher identity diffusion and several psychopathological symptoms such as depression or anxiety (see Table 7). They also were diagnosed more often with a Cluster C disorder and showed higher scores in all interpersonal subscales. However, there was no significant difference in anger or aggression.

26 Appendix 5 (Dammann et al., in preparation)

Table7:
Group comparison of higher versus lower interpersonal problems[27]

	*Higher interpersonal problems (*n*=15)*	*Lower interpersonal problems (*n*=15)*	
IIP, mean (*SD)*			
Total Score	2.25 (0.24)	1.47 (0.32)	t= -7.58, p=0.000**
Domineering/Controlling	7.00 (5.40)	5.60 (3.20)	n.s.
Vindictive/Self-Centred	10.88 (5.80)	9.53 (4.19)	n.s.
Cold/Distant	15.07 (5.27)	10.87 (5.02)	t= -2.23, p=0.034*
Socially Inhibited	21.67 (4.61)	15.00 (6.21)	t= -3.34, p=0.002**
Non-assertive	24.27 (6.05)	13.60 (6.69)	t= -4.58, p=0.000**
Overly Accommodating	23.20 (4.54)	12.73 (4.48)	t= -6.36, p=0.000**
Self-Sacrificing	25.93 (3.60)	16.31 (4.63)	t= -6.35, p=0.000**
Intrusive/Needy	16.27 (4.13)	10.68 (4.47)	t= -3.56, p=0.001**
IPO, mean (*SD)*			
Primitive defences	45.20 (7.97)	40.92 (9.33)	n.s.
Identity diffusion	67.27 (11.45)	57.87 (12.89)	t= -2.11, p=0.044*
Reality testing	43.20 (13.74)	38.87 (11.75)	n.s.
Aggression	33.96 (9.02)	33.67 (7.42)	n.s.
Moral values	24.60 (7.40)	25.80 (7.95)	n.s.
SCL-90-R, mean *(SD)*			
Global severity index	1.83 (0.50)	1.14 (0.48)	t= -3.85, p=0.001**
BDI, mean *(SD)*			
Depression Score	30.98 (7.22)	23.89 (9.70)	t= -2.27, p=0.032*
STAI, mean *(SD)*			
State Anxiety	62.00 (9.02)	52.27 (9.53)	t= -2.87, p=0.008**
Trait Anxiety	62.93 (7.58)	55.17 (7.05)	t= -2.90, p=0.007**
STAXI, mean *(SD)*			
State Anger	17.34 (6.15)	19.19 (8.67)	n.s.
Trait Anger	20.60 (7.39)	23.67 (7.51)	n.s.
Cluster C Disorder, n (%)			
Yes	13 (86.7)	7 (46.7)	χ^2= 5.40, p=0.020*
No	2 (13.3)	8 (53.3)	

*Notes. SD= standard deviation, n.s.= non-significant, * p<0.05, ** p<0.01*

After 12 weeks of inpatient treatment patients reported a decrease in all scales except for the IIP subscale Domineering/Controlling (see Table 8). The reduction was significant for the IIP general scale and several subscales, primarily in the submission dimension. Furthermore there was a significant decline in identity diffusion, depression and anger.

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Table 8:
Pre-post-tests of patients receiving disorder-specific treatment (n=30)[28]

Variable	*Mpre*	*SDpre*	*Mpost*	*SDpost*	*t*	*p*
IIP						
Total Score	1.86	0.48	1.66	0.47	3.10	0.004**
Domineering/Controlling	6.30	4.42	7.03	4.92	-1.21	0.235
Vindictive/Self-Centred	10.21	5.02	8.67	4.69	2.13	0.042*
Cold/Distant	12.97	5.49	12.20	5.01	0.78	0.443
Socially Inhibited	18.33	6.35	16.21	5.49	2.26	0.031*
Non-assertive	18.93	8.29	16.60	7.93	3.30	0.003**
Overly Accommodating	17.97	6.93	15.73	6.82	2.35	0.026*
Self-sacrificing	21.12	6.37	17.75	5.93	4.46	0.000**
Intrusive/Needy	13.47	5.09	12.23	4.56	1.84	0.077
IPO						
Identity diffusion	62.57	12.90	58.27	14.20	2.91	0.007**
Primitive Defences	43.06	8.80	41.82	10.81	1.00	0.324
Reality testing-	40.41	12.51	39.51	13.97	0.63	0.536
Aggression	33.81	8.12	33.09	8.08	0.71	0.481
Moral values	25.20	7.58	24.46	6.70	0.75	0.461
BDI						
Depression Score	27.44	9.15	21.68	12.21	3.46	0.002**
STAI						
State Anxiety	57.14	10.38	54.01	12.36	1.26	0.216
Trait Anxiety	59.05	8.20	57.34	9.79	1.29	0.204
STAXI						
State Anger	18.27	7.45	14.53	5.34	3.35	0.002**
Trait Anger	22.13	7.48	20.42	6.36	1.94	0.062
SCL-90						
Global Severity Index	1.48	0.60	1.37	0.67	1.16	0.255

*Notes. M= Mean, SD= standard deviation, * p<0.05, ** p<0.01*

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Chapter 4 - Discussion

The purpose of the BABIS–project was to determine more thoroughly the construct of borderline personality disorder and its several areas of dysfunction with the aim to detect specific borderline subtypes.

In summary, we did not succeed in defining any clear delimitable subgroups of BDP, neither in comorbidity with atypical depression[29] nor concerning identity diffusion[30] nor regarding interpersonal problems[31]. It seems more likely that we have found a range of severity for BPD that manifests itself in a respective range of gradually higher scores in all the diagnostic criteria. The only discrepancy the results showed was for anger and aggression that were significant in one study but not in the two others. This could be due to the fact that we didn't use the same statistical procedure to separate the patients into their respective groups in all papers.

The second goal of our study was to investigate the effects of a 12-week disorder-specific inpatient treatment on borderline patients compared to treatment-as-usual. This comparison turned out to be most difficult, since the TAU patients had been treated in different wards of the clinic with variable durations of stay and had received a lower treatment dose on average. Hence the originally intended matched-controlled study was not feasible.

I will therefore exclusively address in more detail the results of the DST group. After 12 weeks DST patients showed a significant reduction in identity diffusion, depression and anger[32]. As mentioned before[33], previous studies demonstrated effects of DBT on depressive symptoms in addition to reduction of self-harm while beneficial effects on anger, impulsivity and personality organisation were found for TFP. Our study was able to demonstrate these effects for a treatment that combines DBT with TFP modules[34] although obviously we cannot say which part of improvement is due to which part of the treatment. But the study seems to indicate that this combination of DBT and TFP elements has a favourable effect on the course of the disorder.

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29 Appendix 2 (Gremaud-Heitz et al., 2014)

30 Appendix 3 (Sollberger et al., 2012)

31 Appendix 5 (Dammann et al., in preparation)

32 Appendix 4 (Sollberger et al., 2014)

33 see section 3.3.

34 self-harm wasn't measured in our study

We would have been very interested in investigating whether these effects at the end of treatment could be maintained for 6 and 12 months respectively after the patients left the clinic. We concluded that – unfortunately – our dropout rate was too high for this kind of investigation. Interestingly, the drop-out rate in the DST-group was lower almost by a factor of 2. This could be due to the fact that the study had been integrated in the DS-treatment and therefore patients and staff felt more strongly connected with it. But borderline patients are generally characterized by high drop-out rates which matches the symptomatic of the disorder (e.g. instability in relationships, mood lability).
Yeomans et al. (1994) found that the therapists' contribution to the contract and to the alliance as well as the patients impulsivity had an impact on the drop-out rate.
Of course there are several limitations of the results of our study. The sample was rather small, especially at the end of treatment for the TAU-group; only inpatients were investigated and we cannot be sure whether the improvement after DST would not also be verifiable after ambulatory treatment. Also the disorder in inpatients is generally more pronounced than it tends to be with out-patients and hence we did not investigate the disorder over its entire spectrum of severity; finally, we didn't include the influence of the patients' medication and were not able to exclude the possible influence of the generally numerous comorbid diagnoses in our borderline patients.
There is still much to find out about BPD and further studies with bigger samples and specific control groups (e.g. with other PD's or depressive disorders) were desirable. Since the DST-specialized ward of the UPK treats not only borderline patients but also patients with narcisstic personality disorder or eating disorders it would be interesting to investigate the effects of DST on these disorders.
In my point of view, future research should, aside from research regarding cause and treatment outcome, concentrate on the question whether BPD could be classified according to levels of severity or if there are certain clusters in combination with comorbid disorders.

Chapter 5 - References

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Chapter 6 - Appendix

1. Article

Gremaud-Heitz, D., Stewart, J. W., & Dammann G. (2011). Das Konzept der atypischen Depression und deutsche Version der "Atypical Depression Diagnostic Scale (ADDS)". *Swiss Arch Neurol Psychiatry, 162,* 148-154.

2. Article

Gremaud-Heitz, D., Riemenschneider, A., Walter, M., Sollberger, D. Küchenhoff, J., & Dammann, G. (2014). Comorbid atypical depression in borderline personality disorder is common and correlated with anxiety-related psychopathology. *Comprehensive Psychiatry, 55*, 650-656.

3. Article

Sollberger, D., Gremaud-Heitz, D., Riemenschneider, A., Küchenhoff, J., Dammann, G., & Walter, M. (2012). Associations between identity diffusion, Axis II disorder, and psychopathology in inpatients with borderline personality disorder. *Psychopathology, 45*, 15–21.

4. Article

Sollberger, D., Gremaud-Heitz, D., Riemenschneider, A., Agarwalla, P., Benecke, C., Schwald, O., et al. (2014). Change in Identity Diffusion and Psychopathology in a Specialized Inpatient Treatment for Borderline Personality Disorder. *Clinical Psychology and Psychotherapy* DOI: 10.1002/cpp.1915

5. Article

Dammann, G., Riemenschneider, A., Walter, M., Sollberger, D., Küchenhoff, J. , Gremaud-Heitz, D. (2015). The Impact of Interpersonal Problems in Borderline Personality Disorder Inpatients on Psychopathology and Treatment Outcome. *In preparation*

Konzept der atypischen Depression und deutsche Übersetzung der «Atypical Depression Diagnostic Scale (ADDS)»

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Summary

Concept of the atypical depression and German translation of "Atypical Depression Diagnostic Scale (ADDS)"

Atypical depression is, contrary to its name, a common disorder that to this day is nosologically not clearly classifiable, which affects some 30% of unipolar depressive patients and is characterised by depressive mood, emotional reactivity, increased sleep, eating disorders and somatic impairment. Atypical depression occurs more often in women than men, and has an earlier onset and a more chronic course of illness than endogenous depression. Comorbidities are common in patients with atypical depression: besides anxiety disorders, other axis I diseases such as addiction, eating or somatoform disorders are mentioned, as well as an association with borderline or cluster C personality disorders.

Despite the early findings it was not until 1994 that atypical depression was included as a specifier of major depressive episodes in DSM-IV. Controversy persists concerning the validity of both the construct and the DSM-IV criteria, especially the need for "mood reactivity" criteria.

This paper contains the German version of the "Atypical Depression Diagnostic Scale (ADDS)", which, since it investigates the criteria in greater detail, is regarded as the most highly elaborated instrument for the diagnosis of atypical depression.

In a short review we demonstrate that, on the basis of several factors – biology, course of illness and treatment response – atypical depression can be considered a separate depressive group.

In summary, the concept of atypical depression is highly relevant, and additional studies investigating the validity and relevance of treatment would therefore be preferable. With regard to DSM-V, a revision of the criteria would seem to be appropriate.

Key words: atypical depression; diagnostic scale; measurement; mood reactivity; nonmelancholic depression; validity

Einleitung

Atypische Depression ist nicht, wie es der Begriff implizieren könnte, ein seltenes klinisches Phänomen, sondern vielmehr eine relativ häufige Form der Depression, die etwa 30% der unipolar depressiven ambulanten Patienten betrifft [1]. Das Attribut «atypisch» bezieht sich dabei auf die ungewöhnlichen klinischen Merkmale dieser Depressionsart, weshalb sie leicht übersehen wird, was oft eine Chronifizierung bis hin zur Suizidalität zur Folge hat. Atypisch depressive Patienten zeichnen sich im Vergleich zu melancholischen Patienten aus durch eine Aufhellbarkeit der Stimmung, Hypersomnie und Hyperphagie, bleierne Schwere des Körpers sowie eine lang anhaltende Überempfindlichkeit gegenüber Zurückweisung.

Das Konzept der atypischen Depression ist von grossem Interesse durch seine Verbindung mit dem weiblichen Geschlecht. Es hat sich gezeigt, dass diese Form in einem bedeutenden Ausmass die geschlechtsspezifischen Unterschiede einer Depression erklären kann [2]. Atypische Depression ist zwei bis dreimal wahrscheinlicher bei Frauen als bei Männern, das Erstauftreten erfolgt in einem jüngeren Alter als bei der endogenen Depression, und der Verlauf ist eher chronisch als episodisch [3].

Der folgende Überblick basiert auf einer Recherche der aktuellen Literatur mittels Pubmed, PsycInfo und Web of Science.

Historische Entwicklung des Konzepts «atypische Depression»

Die Entwicklung des Konzepts begann in den späten 1950er Jahren mit den Untersuchungen von West und Dally in London, die eine Gruppe von Patienten beschrieben hatten, welche nicht auf die Behandlung mit Trizyklika oder Elektrokrampftherapie reagierten, hingegen auf den Monoaminoxidase (MAO)-Hemmer Iproniazid ansprachen [4]. Die Autoren berichteten, dass Iproniazid besonders günstig bei denjenigen Patienten wirke, die «eine Art atypischer Zustände haben, welche zeitweise an ängstliche Hysterie mit sekundärer Depression erinnerten» (pp. 1491).

Diese Patienten unterschieden sich von der typischen depressiven Vergleichsgruppe in erster Linie durch fehlende Merkmale endogener Depression und dem Vorhandensein multipler Phobien und Ängste. West und Dally beschrieben damals jedoch noch keine reversiblen vegetativen Symptome. Dieses Phänomen wurde erst zehn Jahre später von Klein und Davis aufgenommen, die diese Form der Depression besonders bei jüngeren Frauen ohne Melancholie beschrieben.

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Klein und Davis [5] berichteten von Patientengruppen mit einem spezifischen Ansprechen auf MAO-Hemmer und stellten fest: «So genannte ‹atypische Depressionen› kommen bei Patienten mit depressiver Stimmung vor, die die üblichen Konsequenzen der klassischen Depression umkehren und/oder Hypersomnie, Hyperphagie, ansteigende Libido oder Gewichtszunahme zeigen, oder die vorrangig phobisch-ängstliche Tendenzen haben» (pp. 182).

Quitkin und Mitarbeiter [6] formulierten dann die erste operationale Definition der atypischen Depression, welche Stimmungsschwankungen, begleitet von übermässigem Schlaf, Energieverlust, Gewichts- oder Appetitzunahme sowie interpersonelle Sensitivität bei Zurückweisung mit einschloss. In den 1980er Jahren wurde der Ausdruck «atypische Depression» dann jedoch ziemlich variabel verwendet bis das Konzept 1994 dann ins DSM-IV, jetzt DSM-IV-TR [7] aufgenommen wurde mit den folgenden Kriterien A: affektive Reagibilität und B: mindestens zwei von vier Symptomen (Appetit- oder Gewichtszunahme, Hypersomnie, bleierne Schwere des Körpers und lang anhaltende Überempfindlichkeit gegenüber Zurückweisung).

Epidemiologie und Prävalenz

Epidemiologische Studien zeigen, dass atypische Depression relativ häufig in der Allgemeinbevölkerung vorkommt, mit Raten zwischen 0,7 und 4% [2, 8]. In der klinischen Population von depressiven Patienten, die eine Behandlung aufsuchen, finden sich Anteile von 16 bis 36% [9, 10]. Unter depressiven ambulanten Patienten finden sich je nach Studie 15 bis 47%, welche die atypischen Kriterien erfüllen [11–13].

In verschiedenen grossen Studien [3, 13, 14] zeichneten sich Patienten mit atypischen Merkmalen aus durch ein früheres Auftreten depressiver Episoden und eine hohe Behandlungsrate. Sie hatten grössere funktionelle Beeinträchtigungen und – mit einer grösseren Wahrscheinlichkeit – einen chronischen Krankheitsverlauf als Patienten mit nicht atypischer Depression.

Beim Erkrankungsalter belegen zahlreiche Studien, dass die Patienten mit einer atypischen Depression bei der Ersterkrankung anderthalb bis zwei Jahre jünger sind als die nicht atypisch depressiven Patienten [2, 3, 15]. Auch beim Geschlecht scheint ein deutlicher Unterschied zu bestehen, in mehreren Studien findet sich sogar ein Verhältnis zwei zu eins [3, 12, 13].

Komorbiditäten

Komorbiditäten, insbesondere zu Störungsbildern, die früher als neurotisch bezeichnet wurden (z.B. Phobien, Zwangsstörungen), finden sich häufig bei atypisch depressiven Patienten. Sie scheinen auch signifikant häufiger aufzutreten im Vergleich zu nicht atypisch depressiven [15, 16]. Eine Verbindung zu den Angststörungen ist naheliegend, hatten doch West und Dally [4] schon von ängstlicher Hysterie geschrieben, und auch Klein und Davies [5] beschrieben phobisch-ängstliche Tendenzen. Diese Tendenz konnte auch in verschiedenen Studien nachgewiesen werden: So fanden sich signifikant häufiger Kriterien einer Panikstörung [8, 14], höhere Raten sozialer Phobie [3, 15] sowie eine grössere Komorbidität mit verschiedenen Symptomen von Angststörungen [2, 12] bei Patienten mit atypischer Depression.

Bei atypisch depressiven Patienten finden sich aber auch Lebenszeitkomorbiditäten mit Essstörungen [3, 14], somatoformen Störungen [8, 11] sowie Erkrankungen aus dem Suchtbereich [8, 15]. Auch ist die Wahrscheinlichkeit einer komorbiden Achse-II-Störung signifikant erhöht [13, 16], neben einer Verbindung mit der Borderline Persönlichkeitsstörung sind auch Persönlichkeitsstörungen aus dem ängstlichen Cluster C (nach DSM-IV-TR [7]) häufig zu finden [13]. Atypische Depression scheint häufiger auch bei stationären Patienten aufzutreten als früher vermutet [10].

Verbindung mit bipolarer Störung

Oft wird auch von einer starken Verbindung der atypischen Depression mit bipolaren Störungen berichtet, insbesondere mit der Bipolar-II-Störung [2, 17], die bestimmt wird durch eine oder mehrere Episoden einer Major Depression und mindestens einer hypomanen Episode. In einer grossen klinischen Versuchsreihe in Italien [14] zeigte sich, dass Patienten mit einer Bipolar-II-Störung mit einer doppelt so grossen Wahrscheinlichkeit eine atypische Depression aufwiesen als Patienten mit einer unipolaren Depression. Keinen Zusammenhang zwischen Hinweisen auf Bipolarität in der Vorgeschichte und atypischen Merkmalen der Depression fanden hingegen Seemüller und Mitarbeiter [11] in ihrer Studie bei 1073 stationären Patienten.

Therapie der atypischen Depression

Pharmakotherapie

Da die Entwicklung des Konzepts auf dem besonders guten Ansprechen der Patienten auf einen MAO-Hemmer beruht, ist es naheliegend, dass diese Medikamentengruppe als Therapie der Wahl bestanden hat, zumal sie sich im Vergleich mit trizyklischen Antidepressiva als überlegen erwies [18, 19].

Paradoxerweise verloren die MAO-Hemmer allerdings relativ bald nach der offiziellen Aufnahme der atypischen Depression ins DSM-IV ihre alltagspraktische Bedeutung durch die Einführung der bald allgemein die Verschreibungspraxis dominierenden selektiven Serotonin-Wiederaufnahmehemmer (SSRIs) in den 1990er Jahren [20]. Die Bedeutung der MAOIs ging möglicherweise auch deshalb etwas zurück, weil die neueren (reversiblen) Substanzen (insbesondere Moclobemid), die weniger strenge diätische Massnahmen beanspruchen, sich zwar wegen ihrer Sicherheit durchsetzen, aber weniger wirksam erscheinen als die ersten MAOIs wie Phenelzin [21].

Dennoch kommen Stewart [22] oder Thase [20] in neueren Übersichtsarbeiten zur Behandlung zu der Schlussfolgerung, dass MAO-Inhibitoren weiterhin Mittel der ersten Wahl bei der atypischen Depression sein sollten. In den USA erfährt die (ohne sehr strenge diätetische Vorschriften mögliche) transdermale Applikation des ersten selektiven MAO-B Inhibitors Selegilin (EMSAM®, das zuvor in der Parkinson-Behandlung eingesetzt wurde) [23] gegenwärtig eine Renaissance.

Tabelle 1
Diagnosekriterien der atypischen Depression nach DSM-IV-TR [7].

Kriterium A	Affektive Schwingungsfähigkeit (d.h., die Stimmung hellt sich bei aktuellen oder möglichen positiven Ereignissen auf)
Kriterium B	Mindestens zwei der folgenden Merkmale sind erfüllt: 1. Bedeutsame Gewichtszunahme oder Zunahme des Appetits 2. Hypersomnie (Vermehrter Schlaf, mehr als 10 Std./Tag) 3. Bleierne Schwere in Armen und Beinen 4. Überempfindlichkeit gegenüber Zurückweisung (Weit zurückreichendes Verhaltensmuster von Empfindlichkeit gegenüber Zurückweisung durch andere, das zu einer bedeutsamen sozialen oder beruflichen Beeinträchtigung führt)
Kriterium C	Weder die Kriterien für den melancholischen noch für den katatonen Subtypus sind erfüllt

Eine weitere Substanz, die international relativ häufig zur Behandlung der atypischen Depression eingesetzt wird [24], ohne dass jedoch eine prospektive Studie vorliegt, ist das Bupropion (Wellbutrin®), welches auch zum Nikotinentzug verwendet wird. In einer anderen Studie konnte gezeigt werden, dass die Behandlung mit Chrom (600 µg/Tag) im Unterschied zu Plazebo einen signifikanten Einfluss auf das Kohlenhydrat-Craving, den vermehrten Appetit und das vermehrte Essen bei Patienten mit atypischen Depressionen, hatte [25]. Unklar bleibt dabei, ob z.B. bei höherer Dosierung auch die depressive Stimmungslage beeinflusst werden könnte.

Psychotherapie

Bisher finden sich nur wenige Studien, die Psychotherapie bei atypischer Depression verglichen haben.

Jarrett und Mitarbeiter [26] fanden in einer Plazebo-kontrollierten Studie, dass sowohl Phenelzin wie kognitive Psychotherapie der Gabe von Plazebo hochsignifikant überlegen war. Zwischen den beiden Behandlungsverfahren (Phenelzin und KVT) dagegen fanden sich keine Unterschiede in allen gemessenen Items. Beide Therapien erscheinen somit wirksam zu sein, allerdings fanden sich in beiden Gruppen über 40% Non-Responder. Die Studie wurde jedoch wegen ihrer hohen Zahl von eingeschlossenen Patienten und den wenig geeigneten psychometrischen Tests (HRSD-21) kritisiert [27].

In einer interessanten Studie, die möglicherweise Hinweise auf unterschiedliche neurobiologische Beeinflussbarkeit für Psychotherapie von Endophänotypen liefern könnte, fanden Lehto und Mitarbeiter [28], dass von 19 Patienten, die wegen Depression ein Jahr lang psychodynamische Psychotherapie erhalten hatten, nur die Untergruppe mit atypischen Merkmalen (n = 8) in der Single-Photon-Emissions-Computertomographie (SPECT) nach Psychotherapie bei der Dichte der Mittelhirn-Serotonin-Transporter (SERT) eine signifikante Zunahme zeigten.

Die Diagnose «atypische Depression»

Der Begriff «atypische Depression» ist lange Zeit sehr variabel verwendet worden. Erst 1994 etablierte die American Psychiatric Association [7] im DSM-IV die atypische Depression als separaten Subtypen der affektiven Erkrankungen und legte die Diagnosekriterien fest (Tab. 1).

Das DSM-IV orientierte sich bei der Diagnose stark an der von Stewart und Mitarbeitern entwickelten *Atypical Depression Diagnostic Scale* (ADDS) [29], wobei die Kriterien insbesondere bei der affektiven Schwingungsfähigkeit im Gegensatz zur ADDS ungenauer formuliert wurden, weshalb das Eingangskriterium A besonders umstritten ist und als nicht essentiell erscheint [10, 13, 30].

In einer aktuellen Literaturübersicht von Stewart und Mitarbeitern [31] schlagen die Autoren vor, einen frühen Krankheitsbeginn sowie einen chronischen Krankheitsverlauf in die Definition einer atypischen Depression einzubeziehen.

In der in den deutschsprachigen Ländern gültigen ICD-10-Klassifikation ist die atypische Depression nicht als eigenständige Diagnose aufgeführt und muss unter F32.8. (sonstige depressive Episode) kodiert werden [10].

Die «Atypical Depression Diagnostic Scale (ADDS)» von Stewart und Mitarbeitern

Die Columbia-Gruppe begann 1979 mit einer Reihe von Behandlungsversuchen, um zu testen, ob eine prospektiv definierte Population vorzugsweise auf Phenelzin im Vergleich zu Imipramin reagieren würde.

Basierend auf der Literatur sowie aufgrund von Erfahrungen im Klinikalltag definierten sie operationalisierte Kriterien für eine Patientengruppe, bei der sie voraussagten, dass sie überwiegend auf ein MAOI im Gegensatz zu einem TCA ansprechen würde. Einschlusskriterium für die Teilnahme an der Studie war die Diagnose einer depressiven Erkrankung nach DSM-III. Weiter mussten die Patienten fähig sein, zumindest in einem gewissen Grad durch ein positives Ereignis aufgeheitert zu werden. Zusätzlich mussten noch zwei der Symptome «Hypersomnie, bleierne Paralyse, Hyperphagie und pathologische Empfindlichkeit gegenüber Zurückweisung» erfüllt sein.

In den Behandlungsversuchen wurde die Hypothese der Columbia-Gruppe bestätigt: So verbesserten sich unterschiedliche atypisch depressive Kohorten mit grösserer Wahrscheinlichkeit nach der Behandlung mit Phenelzin als mit Imipramin. Aufgrund verschiedener Tests konnte die Gruppe feststellen, dass neben der Stimmungsreaktion mindestens eines der zusätzlichen Symptome vorhanden sein musste, damit sich der Behandlungserfolg des MAOIs gegenüber dem TCA zeigte.

Im Anschluss an diese Untersuchungen wurden die vordefinierten Kriterien noch detaillierter beschrieben und zum Teil erweitert, woraus dann die *Atypical Diagnostic Depression Scale* (ADDS – siehe Anhang) der Columbia-Gruppe entstand, welche als das elaborierteste Instrument für die atypische Depression bezeichnet werden kann [29].

Im Gegensatz zum DMS-IV differenziert die ADDS verschiedene Ausprägungsgrade der einzelnen Kriterien. Zudem wird bei der Hyperphagie unterschieden zwischen gesteigertem Appetit, gesteigerter Nahrungsaufnahme und signifikanter Gewichtszunahme, und auch die pathologische Empfindlichkeit gegenüber Zurückweisung wird in verschiedene Problembereiche unterteilt.

Tabelle 2
Gruppenweise Unterschiede zwischen Patienten mit atypischer Depression, undifferenzierter Depression, Melancholie und Kontrollen (nach Stewart et al. [31]).

	Atypisch vs. Kontrollen	Atypisch vs. Undifferenziert	Atypisch vs. Melancholie	Undifferenziert vs. Kontrollen	Melancholie vs. Kontrollen	Undifferenziert vs. Melancholie
McGinn et al. [33]	*	*	*	–	–	NS
Fountoulakis et al. [34]	*	*	*	*	*	NS
Anisman et al. [35]	*	*	–	NS	–	–
Bruder et al. [37]	*	*	*	NS	*	NS
Stewart et al. [41]	–	A	–	–	–	–
Joyce et al. [42]	–	*	*	–	–	NS

Anmerkungen
* Gruppen unterschieden sich signifikant.
NS Gruppen unterschieden sich nicht signifikant.
– eine oder beide Gruppen waren nicht in der Studie eingeschlossen.
A Patienten mit atypischer Depression unterschieden sich signifikant von nicht atypisch depressiven Patienten in ihrer Reaktion auf die Behandlung, die nicht atypische Gruppe war jedoch nicht weiter unterteilt worden in solche mit und ohne Melancholie.

Im Rahmen einer Studie wurde die ADDS von uns ins Deutsche übersetzt. Zur Validierung der wort- und sinngemässen Übersetzung wurde die deutsche Fassung von einem «native speaker» ins Englische zurück übersetzt und abschliessend von Prof. Stewart für gut geheissen. Die ausführliche Version des Interviews in deutscher und englischer Sprache ist bei den Autoren erhältlich.

Tests zur Reliabilität wurden kurz nach Erscheinen der englischen Version durchgeführt, die Daten sind beim Autor (Prof. Stewart) erhältlich. Da die Validität des Interviews von der Validität des Konstruktes selbst herrührt, stellt sich zunächst die Frage, ob die atypische Depression als eigenständiger depressiver Subtypus abgrenzbar ist.

Atypische Depression: ein valider Subtyp?

Die laufende Definition der atypischen Depression gibt immer wieder Anlass zu Diskussionen und wird von verschiedenen Autoren in Frage gestellt [2, 30, 32]. In verschiedenen Studien konnten keine Verbindung der Schwingungsfähigkeit mit den anderen diagnostischen Kriterien der atypischen Depression gefunden werden [11, 13]. Auch ist «bleierne Schwere» relativ ungenau, wird häufig nicht spontan berichtet und meint nicht «fatigue». In einer Studie fanden Riedel und Mitarbeiter [10] bei insgesamt 829 Patienten mit atypischer und nicht atypischer Depressionsart, dass sich beide Gruppen insbesondere in folgenden Items unterschieden, die signifikant häufiger mit atypischer Depression verbunden waren: Somatische Angst, Genitalstörungen, Depersonalisation und Derealisation, paranoide Symptome (inkl. Misstrauen) und interessanterweise auch Schuld. Kein Unterschied zeigte sich hingegen bei Suizidgedanken.

Stewart und Mitarbeiter haben sich deshalb in einem aktuellen Artikel [33] mit der Validität der atypischen Depression auseinandergesetzt und konnten anhand der nachfolgenden Faktoren aufzeigen, dass die atypische Depression als eigenständiges Konzept bestehen kann.

Biologische Studien

In einer Literaturübersicht zeigt auch Posternak [34] auf, dass verschiedene biologische Kriterien starke Hinweise darauf liefern, dass es sich bei der atypischen Depression um einen eigenen existierenden Subtypus der Depression handle: «Studies involving hypothalamic-pituitary-adrenal axis activity, cerebral laterality, neurochemical profiles, and sleep parameters» (pp.1), siehe Tabelle 2.

McGinn und Mitarbeiter [35] fanden in ihrer Studie unterschiedliche kortikale Reaktionen auf Desipramin bei depressiven Patienten mit atypischen Merkmalen im Vergleich zu anderen depressiven Patienten. Fountoulakis und Mitarbeiter [36] konnten einen veränderten zerebralen Blutfluss bei atypisch Depressiven zeigen, der sich nicht nur von einer gesunden Kontrollgruppe, sondern auch von melancholischen und undifferenzierten depressiven Patienten unterscheidet.

Weitere Studien, welche Kortisol [37], evozierte Potenziale [38], perzeptuelle Verarbeitung [39] sowie Variationen in der Hypothalamus-Hypophysen-Achse [40] evaluieren, liefern ebenfalls deutliche Hinweise, dass Personen mit atypischer Depression neurobiologisch von anderen Depressionsgruppen unterschieden werden können.

Krankheitsverlauf

Bereits die Columbia-Gruppe [29] berichtete von einem früheren Erstauftreten und einem chronischeren Verlauf bei atypisch depressiven im Vergleich mit melancholischen Patienten. Ein Resultat, welches auch durch grosse epidemiologische Studien gefestigt wird [2, 15].

Der Krankheitsverlauf kann auch beschrieben werden durch eine Bestimmung der Konstanz der Symptome depressiver Subtypen über Zeit. Nierenberg und Mitarbeiter [41] wie auch Kendler und Mitarbeiter [42] fanden in ihren Studien, dass bei einer späteren Befragung über 70% der Patienten die gleichen Symptome aufwiesen oder berichteten wie anfänglich. So scheinen vor allem die neurovegetativen Symptome über Zeit stabil zu sein.

Pharmakologische Differenzierung

Stewart und Mitarbeiter [43] konnten eine signifikante Dreifachinteraktion zwischen Behandlung (Imipramin vs. Plazebo), Subtyp (atypisch vs. nicht atypisch) und Behandlungserfolg aufzeigen. Dabei fand sich bei atypisch Depressiven keine starke Verbesserung mit dem Medikament im Vergleich zum Plazebo, während die nicht atypisch depressiven Patienten einen stabilen Imipramineffekt aufwiesen.

In der Studie von Joyce und Mitarbeiter [44] wurden 195 depressive Patienten randomisiert einer Behandlung mit Nortriptylin und Fluoxetin zugeteilt. Dabei waren beide Medikamente gleich wirksam bei melancholischen und undifferenzierten depressiven Patienten, während sich bei den atypisch depressiven eine signifikante Überlegenheit des Fluoxetins zeigte.

Fazit

Diese Resultate sprechen deutlich dafür, dass Patienten, welche die DSM-IV-Kriterien einer atypischen Depression erfüllen, sich von Patienten mit melancholischer und undifferenzierter Depression unterscheiden, und die atypische Depression als eigener Subtypus abgrenzbar ist.

Trotzdem gibt die laufende Definition der atypischen Depression immer wieder Anlass zu Diskussionen und wird von mehreren Autoren in Frage gestellt [2, 30]. Insbesondere die hierarchische Position des Kriteriums «affektive Schwingungsfähigkeit» wird immer wieder diskutiert, zumal einerseits in verschiedenen Studien keine Verbindung mit den anderen diagnostischen Kriterien der atypischen Depression gefunden werden konnte [12, 35]. Andererseits zeigte sich, dass die Schwingungsfähigkeit auch bei nicht atypischen Depressionen häufig vorhanden ist [10]. Es ist bis jetzt jedoch noch keine Studie bekannt, die ausschliesslich eine depressive Patientengruppe ohne affektive Schwingungsfähigkeit untersucht hat. Einige Autoren schlagen zudem vor, weitere Kriterien (z.B. somatische Symptome, Chronizität) in die Diagnose aufzunehmen [29, 45].

Eine Überarbeitung der momentan geltenden Kriterien im Hinblick auf das DSM-V scheint demnach angebracht. Die Autoren dieses Artikels regen an, die Diagnose atypische Depression um die Kriterien «früher Krankheitsbeginn» (<20 J) sowie «deutliche Chronizität» (nicht länger als zwei Monate andauerndes Wohlbefinden) zu erweitern, da diese eine gute Vorhersagbarkeit für den Therapieverlauf zeigten [31].

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Anhang

Atypische Depression: Diagnostik-Skala (ADDS)[1]

I. DSM Depression

Der Patient erfüllt die Kriterien einer Affektiven Störung gemäss DSM.

II. Reaktionsfähigkeit der Stimmung

Ausmass, in welchem positive umgebungsbedingte Ereignisse verknüpft sind mit einer Verbesserung der Stimmung (Kriterium erfüllt, wenn maximale Reaktion ≥50%).
a) übliche Reaktion → %
b) maximale Reaktion → %

III. Assoziierte Eigenschaften

Zwei Kriterien von A bis D müssen erfüllt sein.

A: Schlaf
Exzessives Schlafen (mind. drei Tage in der Woche zehn Stunden oder mehr).

B: «Bleierne Paralyse»
Das körperliche Empfinden, sich schwer, bleiern oder von Gewichten heruntergezogen zu fühlen. Kriterium erfüllt bei einem Rating von 4 bis 6.
1. Adäquate oder übermässige Energie
2. Milde Abnahme von fragwürdiger Signifikanz wenn depressiv
3. Mässige Abnahme mit möglicher funktionaler Beeinträchtigung, aber keine oder seltene bleierne Paralyse wenn depressiv
4. Merkliche Abnahme; bleierne Gefühle mindestens eine Stunde im Tag für mindestens drei Tage in der Woche wenn depressiv
5. Ernsthafte Abnahme; bleierne Gefühle die meiste Zeit an den meisten Tagen wenn depressiv
6. Extreme Abnahme; bleiernes Gefühl den grössten Teil der Zeit oder immer wenn depressiv

C: Appetit/Gewicht
Kriterium erfüllt bei einem Rating von 4 bis 6 in C1, C2 oder C3.

C1: Appetit
Übermässiger Drang zu Essen (unabhängig von der konsumierten Menge).
1. Kein gesteigerter Appetit
2. Leichte Zunahme von fragwürdiger Signifikanz
3. Minimale Zunahme; Appetit war ein wenig gesteigert wenn depressiv (z.B. will gelegentlich bingen oder mehrmals in der Woche einen Extraimbiss oder eine zweite Portion)
4. Merkliche Zunahme; will mindestens drei Mal in der Woche bingen oder hat an den meisten Tagen (5/7) den starken Drang, sich zu überessen
5. Ernsthafte Zunahme; hat den Drang, an den meisten Tagen zu bingen oder will sich die meiste Zeit überessen
6. Extreme Zunahme; will praktisch den ganzen Tag bingen

C2: Essen
Exzessives Konsumieren von Nahrung.
1. Keine Störung; isst normal oder zu wenig wenn depressiv
2. Minimale Zunahme; kann gelegentlich einen Happen essen wenn depressiv, dies scheint aber nicht klinisch signifikant zu sein
3. Leichte Zunahme; z.B. isst eine Zwischenmahlzeit oder andernfalls überisst sich wenn depressiv, dies aber in einem leichten Ausmass
4. Mässige Zunahme; z.B. bingt zweimal in der Woche oder isst beinahe täglich Zwischenmahlzeiten, oder nimmt mehrmals im Tag an drei Tagen der Woche einen Imbiss zu sich wenn depressiv
5. Ernsthafte Zunahme; z.B. bingt mindestens vier Mal in der Woche oder nascht praktisch dauernd wenn depressiv
6. Extreme Zunahme; bingt täglich wenn depressiv

C3: Gewicht

Signifikante Gewichtszunahme.

1. Keine Gewichtszunahme wenn depressiv
2. Minimale Gewichtszunahme von fragwürdiger Signifikanz (0–4 Pfund)
3. Leichte Gewichtszunahme, weniger als 10 Pfund
4. Mässige Gewichtszunahme, 10–15 Pfund
5. Markante Gewichtszunahme, 15–20 Pfund
6. Extreme Gewichtszunahme, mehr als 20 Pfund

D: Empfindlichkeit gegenüber Ablehnung/Zurückweisung/ Kritik

Kriterium erfüllt bei einem Rating von 46 in D1, D2, D3, D4 oder D5.

D1: Zwischenmenschliche Empfindlichkeit

Emotionale Überreaktion auf Zurückweisung oder Kritik.

1. Keine; geht locker um mit den Kurven des Lebens
2. Minimal; fühlte sich ein wenig verletzt oder ist ein wenig gekränkt
3. Mild; ist etwas entmutigt oder wütend, kommt aber rasch wieder auf die Beine
4. Mässig; ist deutlich masslos entmutigt oder wütend
5. Ernsthaft; reagiert deutlich übertrieben über eine längere Zeit
6. Extrem; wird ausserordentlich depressiv oder wütend für längere Zeit oder sucht übertrieben nach Situationen, in denen sie/er schlecht behandelt worden ist

D2: Qualität von Beziehungen

Turbulente oder stürmische Beziehungen aufgrund der Überreaktion auf Zurückweisung oder Kritik.

1. Keine; Beziehungen sind problemlos
2. Minimal; es gibt geringfügige Schwierigkeiten, aber von zweifelhafter Signifikanz
3. Leicht; es gibt etwas Aufruhr in Beziehungen, diese sind aber ziemlich stabil
4. Mässig; Eifersucht oder Empfindlichkeit gegenüber Kritik resultiert in stürmischen Beziehungen, Schwierigkeiten, den Beruf aufrechtzuerhalten oder einen Haushalt zu führen
5. Ernsthaft; häufige Streitereien, Missverständnisse, Auseinandersetzungen aufgrund der Empfindlichkeit gegenüber Zurückweisung/Ablehnung oder Kritik
6. Extrem; zwischenmenschliche Schwierigkeiten wegen der Empfindlichkeit gegenüber Ablehnung oder Kritik sind nahezu konstant

D3: Funktionale Beeinträchtigung

Beeinträchtigung in der Schule oder bei der Arbeit aufgrund der Überreaktion auf Kritik oder Zurückweisung.

1. Keine Beeinträchtigung
2. Minimale; geringfügige Beeinträchtigung, die aber nicht klinisch signifikant ist
3. Leichte; einige leichte Beeinträchtigungen, macht nicht den besten, aber doch gerade noch seinen Job
4. Mässige; z.B. verlässt die Arbeit früher, erfüllt wichtige Hausarbeiten nicht, betrinkt sich als direkte Reaktion auf Kritik oder Zurückweisung mindestens viermal in zwei Jahren
5. Ernsthafte; z.B. hat bei der Arbeit mindestens zwölfmal in zwei Jahren gefehlt wegen Kritik oder Zurückweisung
6. Extreme; Wiederholte funktionale Beeinträchtigung (zu viele zum Zählen)

D4: Vermeidung von Beziehungen

Fehlen von Beziehungen wegen Angst vor Zurückweisung.

1. Keine; ht Beziehungen oder der Beziehungsmangel ist eindeutig nicht wegen der Vermeidung von Zurückweisung
2. Minimale; ist minimal besorgt bezüglich Zurückweisung, hat aber Beziehungen
3. Leicht; ziemlich besorgt bezüglich eventueller Zurückweisung, zwingt sich aber dazu, ein paar Beziehungen zu haben
4. Mässig; vermeidet intime Beziehungen wegen Zurückweisungsangst, erlaubt aber oberflächliche Beziehungen
5. Ernsthafte; hat nur minimalen, oberflächlichen Kontakt zu anderen wegen Vermeidung von Zurückweisung
6. Extreme; absoluter Einsiedler aufgrund der Zurückweisungsangst

D5: Vermeidung anderer Zurückweisung

Vermeidung anderer wichtiger Lebensaufgaben, um einer Zurückweisung zu entgehen.

1. Keine; keine Vermeidung
2. Minimal; minimale Vermeidung wegen Zurückweisungsempfindlichkeit, aber mit minimaler oder keiner Beeinträchtigung
3. Leicht; gewisse Vermeidung aufgrund der Empfindlichkeit gegenüber Ablehnung mit leichter Beeinträchtigung
4. Mässig; signifikante Beeinträchtigung wegen der Vermeidung von Zurückweisung
5. Ernsthaft; merkliche Beeinträchtigung aufgrund der Vermeidung einer möglichen Ablehnung
6. Extrem; z.B. ist unfähig, einen Job zu behalten oder die Schule zu besuchen aus Angst vor einer Zurückweisung; oder ist unfähig, während zweier Jahre mit jemandem Kontakte zu knüpfen

Diagnose Atypische Depression

0 = Nicht depressiv (I nicht erfüllt)
1 = Nicht atypische Depression (II maximale Reaktion <50%)
2 = Einfache Stimmungsreaktionsdepression (II max. Reakt. ≥50%, keines von III A–D ist erfüllt)
3 = Mögliche atypische Depression (II max. Reakt. ≥50%, eines von III A–D ist erfüllt)
4 = Definitive atypische Depression (II max. Reakt. ≥50%, zwei oder mehr von III A–D sind erfüllt)

1 Deutsche Übersetzung: Daniela Gremaud-Heitz und Gerhard Dammann (2008)

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Comorbid atypical depression in borderline personality disorder is common and correlated with anxiety-related psychopathology

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Abstract

Background: The core features of borderline personality disorder (BPD) are affective instability, unstable relationships and identity disturbance. Axis I comorbidities are frequent, in particular affective disorders. The concept of atypical depression is complex and often underestimated. The purpose of the study was to investigate the comorbidity of atypical depression in borderline patients regarding anxiety-related psychopathology and interpersonal problems.

Methods: Sixty patients with BPD were assessed with the Structured Clinical Interviews for DSM-IV Axis I and II Disorders (SCID I, SCID II) as well as the Atypical Depression Diagnostic Scale (ADDS). Additionally, patients completed a questionnaire (SCL-90-R, BDI, STAI, STAXI, IIP-C).

Results: Forty-five BPD patients (81.8%) had a comorbid affective disorder of which 15 (27.3%) were diagnosed with an atypical depression. In comparison to patients with major depressive disorder or no comorbid depression, patients with atypical depression showed significant higher scores in psychopathological symptoms regarding anxiety and global severity as well as interpersonal problems.

Conclusions: The presence of atypical depression in borderline patients is correlated with psychopathology, anxiety, and interpersonal problems and seems to be of clinical importance for personalized treatment decisions.

1. Introduction

Borderline is one of the most common personality disorders that affects about 1% to 2% of the general population, around 10% psychiatric outpatients and 20% psychiatric inpatients. The diagnosis is more common in women (75%) than in men [1,2].

BPD was included in 1980 in the DSM-III classification [3]. The main characteristics include affective instability, unstable relationship patterns, disturbed identity and impulsivity. DSM-IV-TR defines affective instability as intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days, due to a marked reactivity of mood [4].

BPD is considered both psychologically [5] and biologically [6] as a heterogeneous disorder and is associated with high comorbidity [7]. Biological vulnerability and developmental insults combined determine the presentation of BPD. The diagnostic criteria of BPD can be organized into four sectors of psychopathology: affective, cognitive, behavioural and interpersonal criteria [8]. Patients vary widely in their severity of manifestation of these factors and even do not need to be impaired in all four factors. There are 126 different possibilities (clusters) to fulfil the diagnostic criteria for BPD (at least 5 of 9 different criteria) [9]. These dissimilarities can lead to alternate courses of the disorder [10] as well as different treatment responses.

The disorder of affectivity in borderline disorder is conceptualized in different ways. Psychiatrists emphasize either the disorder of affect regulation with difficulty of personality-conditioned affect control [11,12], or the emotional dysregulation due to elevated biological vulnerability [13].

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Gunderson and Phillips [14] point out that depressive disorder in borderline disorders shows a qualitatively different characteristic than in major depression being more developmentally and interpersonally based.

Comorbidities are very common in patients with BPD and seem to predict the characteristic as well as the course of the disorder. Several studies have found that borderline patients are often diagnosed with an Axis I disorder (e.g. anxiety disorders and substance abuse) [8,15]. However the most frequent comorbidities are affective disorders, especially major depression, which occurs in 70% to 90% of all borderline patients [16,17]. Zanarini et al. [17] reported that 80% had experienced a major depression episode at some point in their medical history. It seems that a specific depressive subtype is often connected with BPD; Posternak and Zimmermann [18] found that 27% of their borderline patients had a comorbid atypical depression (AD). The international BRIDGE study [19] examined 2658 patients with an MDD regarding BPD and bipolar disorder. A bipolar diagnosis was more frequent in the non-borderline group whereas borderline patients reported significantly more atypical features.

Atypical depression (AD) was introduced to specify major depressive episodes in DSM-IV following a series of antidepressant trials showing that such patients responded preferentially to monoamine oxidase inhibitors (MAOIs) [20]. This depressive form is characterized by depressive mood, emotional reactivity, increased sleep, eating disorders and somatic impairment and affects about 30% of unipolar depressive patients, mostly women. Biological studies [21] as well as statistical classifications [22] support the hypothesis of a distinct depressive subtype. Compared to melancholia and other depression, atypical depression shows an earlier age of onset and a more chronic course of illness [23]. The quality of the depressive experience in borderline personality disorder has always been perceived to be different from the depression experienced in major depression (MDD) [24].

Perugi and colleagues [25] compared patients who met the DSM-IV criteria for major depressive episode with atypical features in terms of a comorbid BPD. The group with a comorbid borderline disorder had significant higher lifetime comorbidity for bulimia nervosa, cyclothymia and Axis II disorders of the anxious and dramatic cluster (narcissistic, dependent and avoidant). This group also scored higher on multiple Atypical Depression Diagnostic Scale items (mood reactivity, interpersonal sensitivity, functional impairment, avoidance of relationships and other rejection avoidance). Most interestingly, heightened rejection sensitivity seems to be a feature in both AD and BPD [26].

Deliberate self-harm is correlated with heightened sensitivity to interpersonal rejection [27]. High rejection sensitivity is also associated with increased borderline personality features among people low in self-reported executive control and among those high in self-reported executive control, the relationship between rejection sensitivity and borderline personality features is attenuated [28]. Patients with BPD may be more sensitive to rejection, and these fears of rejection may result in increased emotion dysregulation and subsequent behavioral problems [29] or rage [30].

Anxiety disorders seem to be rather common in borderline personality disorder [31,32]. Silverman et al. [33] studied the comorbidity of patients with an Axis II disorder and found rates of 89% anxiety disorders in BPD patients. A national epidemiologic survey with over 34,000 adults [34] also showed a high co-occurrence of anxiety disorder with BPD.

However, AD is also reported to be connected to anxiety disorders. Gili and colleagues [35] compared AD, melancholic and non-melancholic depression in non-borderline patients and found that AD patients had higher rates of comorbid anxiety disorders. More specific studies showed a correlation of AD with social phobia and panic disorder [36–38].

Given that anxiety and rejection sensitivity are common in both AD and BPD the question arises how the co-occurrence of the both disorders is affecting the patient? From our point of view until now there has not been a study investigating BPD and comorbid AD in reference to anxiety.

2. Aims of the study

Since BPD and depression are rather common in co-occurrence the aim of our study was to more closely examine a specific group of depression—atypical depression—in association with BPD. We expected patients with comorbid AD to show a more severe psychopathology compared to other BPD patients with either a different type of depression or no depression at all.

Another hypothesis was whether this co-occurrence of AD leads to more interpersonal problems in BPD patients.

3. Methods

3.1. Study design and participants

All patients were inpatients at the Psychiatric Hospital of the University of Basel and were diagnosed with a borderline personality disorder (BPD) according the DSM-IV-TR criteria. Patients participated in a matched-controlled inpatient study for BPD patients (Basel Borderline Inpatient Study [BABIS]). The aims of this study were to compare the effects of transference focused psychotherapy (TFP)-based disorder-specific inpatient treatment versus treatment as usual and to identify the possible influence of subgroups within the heterogeneous group of BPD patients. Detailed descriptions of the aims, methods and sample characteristics of the Basel Borderline Inpatient Study (BABIS) supported by a research grant from the Swiss National Science Foundation have been reported separately [39].

Exclusion criteria were schizophrenia, schizoaffective disorder, active psychosis or acute manic episode.

Written informed consent was obtained from each patient. The study was approved by the local ethics committee (EKBB).

3.2. Interviews

Clinically experienced interviewers attended a special education of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P) [40] and for DSM-IV Axis II Disorders (SCID-II) [41] and were trained to pay particular attention to distinguishing Axis I mental state conditions from Axis II personality trait phenomena. The SCID I and II are semi-structured interviews for assessing clinical and personality disorders. High interrater reliability has been shown for both interviews [42,43].

Additionally the Atypical Depression Diagnostic Scale (ADDS) [44,45] was used to examine atypical depression more detailed. The ADDS is a semistructured interview designed to investigate the presence and severity of atypical features during current depressive episodes.

3.3. Questionnaire data

To measure the general psychiatric symptoms and subjective complaints, we administered the SCL-90-R [46], the Beck Depression Inventory [47], the Spielberger State and Trait Inventory [48], and the Spielberger State and Trait Anger Inventory [49].

For evaluation of interpersonal criteria we used the Inventory of Interpersonal Problems [50], a 64-item self-report instrument designed to measure interpersonal deficiencies and excesses in eight subscales (e.g. too responsible, too controlling). External validity of the IIP-C scales has been demonstrated.

3.4. Statistical analyses

All statistical analyses were conducted with SPSS/20.0. Assumption of homoscedasticity and normality distribution was checked prior to the analysis. χ^2 Tests were used for testing intergroup differences. Multivariate analysis and one-way ANOVA parametric method were performed for group comparison as well as Student's *t*-test. All statistical tests were considered significant at a two-sided level of $p < 0.05$.

4. Results

Sixty patients diagnosed with borderline personality disorder (BPD) were included in the study and interviewed. Five patients did not complete the questionnaire and were therefore excluded. Of the 55 patients included in the study, 44 (80%) were female, 11 (20%) were male. The mean age was 28.9 years (SD = 8.7) (see Table 1).

Fifty-three patients (96.4%) were diagnosed with a comorbid Axis I Disorder, most frequently with an affective disorder ($n = 45$, 81.8%). Thirty-five patients (63.6%) showed a comorbid Axis II disorder, predominant a Cluster C disorder ($n = 28$, 50.9%). An anxiety disorder was diagnosed in 29 patients (52.7%). Fifteen patients (27.3%) were given the diagnosis of an atypical depression. All patients with comorbid atypical depression met the BPD criterion of affective instability.

Table 1
Demographic and clinical characteristics of the patients.

	Borderline patients ($n = 55$)
Age, mean (SD)	28.86 (8.74)
Gender, *n* (%)	
Female	44 (80)
Male	11 (20)
Marital status, *n* (%)	
Living alone	43 (78.2)
Living with a partner	12 (21.8)
Current employment, *n* (%)	
Employed (full/part time)	28 (50.9)
Unemployed	27 (49.1)
Years of education, *n* (%)	
None	1 (1.8)
<9	23 (41.8)
9–12	21 (38.2)
>12	10 (18.2)
Duration of illness, *n* (%)	
<1 year	4 (7.3)
1–5 years	18 (32.7)
5–10 years	9 (16.4)
10–20 years	18 (32.7)
>20 years	6 (10.9)
Comorbid Axis I disorder, *n* (%)	
None	2 (3.3)
Affective disorder	45 (81.8)
Anxiety disorder	29 (52.7)
Substance related disorder	34 (61.8)
Eating disorder	19 (34.5)
Comorbid Axis II disorder, *n* (%)	
None	19 (34.5)
Cluster A	10 (18.2)
Cluster B	6 (10.9)
Cluster C	28 (50.9)
n/a	1 (1.8)

SD = standard deviation.

To further analyze our results we sub-divided our patients in the following three groups: (1) patients with Atypical Depression, (2) patients with a depression other than AD and (3) patients with no depression.

One-way ANOVA found significances in depression (BDI, $p = 0.002$), anxiety (STAI, state anxiety $p = 0.002$; trait anxiety $p = 0.001$), scales regarding general psychopathology (SCL-90-R, GSI $p = 0.011$), and interpersonal problems (IIP-C, $p = 0.003$).

Furthermore the AD group was diagnosed significantly more often with a comorbid anxiety disorder ($\chi^2 = 0.002$) than the other two groups.

However there were no differences in the three groups regarding anger or aggression.

As Table 2 displays, group 1 (AD) showed the highest scores in the significant data. Independent-measures *t*-test

Table 2
Intergroup differences regarding psychopathology.

	Group 1: atypical depression (n = 15)	Group 2: other depression (n = 30)	Group 3: no depression (n = 10)	F-value (p)
SCL-90-R, mean (SD)				
Global severity index (GSI)	1.7 (0.6)	1.3 (0.7)	1.1 (0.6)	4.936 (0.011)*
Somatization	16.2 (11.4)	11.0 (7.8)	10.0 (5.8)	2.658 (0.080)$^{n.s.}$
Obsessive–compulsive	18.7 (8.4)	13.7 (7.6)	10.1 (4.6)	6.910 (0.002)**
Interpersonal sensitivity	18.1 (7.1)	14.1 (8.4)	11.3 (6.2)	3.890 (0.027)*
Depression	31.0 (10.1)	24.9 (12.4)	18.1 (7.8)	7.092 (0.002)**
Anxiety	18.4 (7.5)	12.1 (7.9)	12.5 (7.4)	3.874 (0.027)*
Hostility	9.1 (5.5)	7.5 (5.4)	6.6 (5.3)	1.022 (0.367)$^{n.s.}$
Phobic anxiety	13.2 (7.5)	7.3 (6.9)	5.7 (5.5)	6.269 (0.004)**
Paranoid ideation	8.7 (4.0)	6.6 (5.3)	6.2 (5.6)	1.342 (0.270)$^{n.s.}$
Psychoticism	11.5 (8.3)	9.5 (7.5)	8.5 (7.1)	0.704 (0.499)$^{n.s.}$
BDI, mean (SD)				
Sum	30.3 (8.5)	25.7 (11.5)	17.5 (9.9)	7.151 (0.002)**
STAI, mean (SD)				
State anxiety	59.9 (11.1)	51.1 (13.9)	48.5 (12.3)	6.961 (0.002)**
Trait anxiety	62.4 (6.9)	55.2 (11.4)	50.5 (11.8)	8.501 (0.001)**
STAXI, mean (SD)				
State anger	20.7 (9.3)	16.0 (5.9)	15.1 (7.1)	2.804 (0.070)$^{n.s.}$
Trait anger	21.9 (6.9)	21.8 (7.7)	19.4 (5.9)	0.773 (0.467)$^{n.s.}$
IIP, mean (SD)				
Sum	2.0 (0.4)	1.8 (0.6)	1.3 (0.6)	6.548 (0.003)**
Domineering/Controlling	5.5 (3.8)	7.6 (4.8)	6.1 (4.9)	0.955 (0.392)$^{n.s.}$
Vindictive/Self-centered	11.2 (3.7)	11.5 (5.6)	10.1 (5.5)	0.380 (0.686)$^{n.s.}$
Cold/Distant	12.5 (5.1)	13.3 (6.3)	11.3 (5.3)	0.562 (0.574)$^{n.s.}$
Socially inhibited	20.1 (6.5)	16.8 (7.1)	12.4 (5.8)	6.440 (0.003)**
Nonassertive	21.5 (8.2)	16.4 (7.1)	9.9 (8.2)	9.924 (0.000)**
Overly accommodating	20.0 (5.9)	15.2 (7.9)	11.5 (5.2)	8.160 (0.001)**
Self-sacrificing	21.8 (5.4)	18.8 (7.7)	14.3 (5.8)	6.476 (0.003)**
Intrusive/Needy	12.5 (4.3)	12.6 (6.6)	9.6 (6.3)	1.537 (0.225)$^{n.s.}$

SD = standard deviation, n.s. = non-significant.
* $p < 0.05$.
** $p < 0.01$.

between the three groups showed that the AD group can be distinguished from the others groups in trait anxiety (STAI), anxiety, obsessive-compulsion, phobia and GSI (SCL-90-R) as well as overly accommodating (IIP-C).

5. Discussion

Our major finding was that patients with an atypical depression showed the highest scores in all psychopathological data. A possible explanation of our result could be summative effects since both conditions BPD and AD are associated with anxiety and interpersonal sensitivity.

This finding is consistent with a study of McGinn et al. [51] which compared major depressive disorder patients with and without atypical depression (AD). AD predicted the presence of comorbid Axis I (100% AD vs. 33% non-AD), Axis II (90% vs. 35%), and both Axis I and II (65% vs. 8.14%) disorders. The high prevalence of Axis I and II comorbidity in major depression might be explained, at least in part, by the presence of atypical depression. Significant differences between the three groups in our study (atypical depression, other depression, no depression) were found in results on depression, general psychopathology, anxiety and interpersonal sensitivity.

Affective disturbances in borderline personality disorder are yet not clearly understood so further studies should continue to deepen our knowledge on different affective disorders in BPD patients (depression, dysthymia, dysphoria, and other form of affective pain). Twenty-five dysphoric states (mostly affects) were found to be significantly more common among borderline patients than controls in the study of Zanarini et al. [31] but nonspecific to borderline personality disorder. Equally important, overall mean Dysphoric Affect Scale scores correctly distinguished borderline personality disorder from other personality disorders in 84% of the subjects. The results of the Zanarini study [31] suggest that the subjective affective pain of borderline patients may be both more pervasive and more multifaceted than previously recognized, and that the overall "amplitude" of this pain may be a particularly good marker for the borderline diagnosis. Consistent with our results (see Table 2: BDI score of AD and other depression) Levy et al. [52] could not find differences between depressed, depressed borderline, and borderline non-depressed inpatients in overall level of impairment or severity of depression. Phenomenologically,

however, depressive experiences were quite different in this study. Subjects with borderline personality disorder, with and without a diagnosed depressive disorder, scored higher than subjects with depression only on the measure of anaclitic neediness (severe emotional dependence on another person, especially relating to the dependence of an infant on a mother or surrogate mother), correlated with interpersonal distress, self-destructive behaviors, and impulsivity.

Mood lability and interpersonal sensitivity traits could be related by a "cyclothymic temperamental diathesis" or "borderline-affective" cluster [25,53]. This cluster, in turn, seems to underlie the complex pattern of anxiety, sensitivity, mood and impulsive disorders which is clinically shared by atypical depression, some bipolar II, few bulimia nervosa [54] and several borderline patients. Cyclothymic reactivity and neurotic features (i.e., atypicality and panic attacks) or structural problems may appear (in line with the description by the French psychiatrist Kahn [55] relevant to the definition of what today is considered bipolar II disorder [56].

In sum our findings could also be suitable with Kernberg's model [57] differentiating biological and characterological depression. In the case of characterological depression, often associated with chronic suicidal tendencies, depressive affect like other affects experienced by the patient, corresponds to the underlying internalized object relations.

A distinct subgroup of borderline patients could be characterized by co-occurrence of AD associated with high phobic and general anxiety, higher general psychopathology and interpersonal problems but no differences concerning psychoticism and paranoid ideation. A possible connecting mechanism for both borderline personality disorder and atypical depression related pathology could be increased rejection sensitivity giving rise to interpersonal problems.

Possible methodological limitations of this study are the small sample size and the fact that all BPD patients (in all of the three groups) were severely disturbed (high psychopathological scores, see Table 2) and had fewer partners and high comorbidity (see Table 1). It remains unclear if a comorbid atypical depression would also be associated with more severe psychopathology, anxiety and interpersonal problems in other personality disorders than BPD.

In summary the fundamental pattern of atypical depression is represented by chronic mild depressions, which are characterized by a younger age at onset, female predominance, interpersonal rejection sensitivity, and mood lability, which are difficult to distinguish from characterological pathology. Patients who present with such patterns are frequently diagnosed with borderline, histrionic, or avoidant personality disorders. Congruent with our results the New South Wales University group (see [58,59]) asserts the structural priority of anxiety symptoms over mood symptoms and the significance of interpersonal rejection sensitivity in atypical depression. This concept overlaps considerably with that of "hysteroid dysphoria," which was proposed by Klein and Liebowitz [60], and was one precursor of Columbia group's later concept of atypical depression.

Differential treatment response of subtypes of patients with borderline personality has been identified [61–63]. A careful phenomenological analysis of early clinical phenotypes, a clinical staging (with valid severity indexes), and strategic biomarker research are the building blocks for a future personalized psychiatry [64]. Current therapies are limited because they do not recognize or accommodate the extensive heterogeneity of borderline personality disorder and its complex etiology [65]. Currently, insufficient evidence is available supporting most personalizing variables for borderline personality disorder or depression (an important exception is cytochrome p450 activity). "Some of the features that have potential as personalizing variables that can help predict response to particular treatments, pending replication studies, include sex, hormonal status, atypical depression, childhood trauma, family history of mental illness, and certain biomarkers and genetic polymorphisms" [66, pp. 1].

Unfortunately the concept of atypical depression has become overextended and gradually lost its construct validity. Therefore, the diagnostic criteria for atypical depression should be reconsidered in reference to various definitions and concepts and refined through accumulated clinical research (see Ohmae [67]). "A fuller appreciation of the BPD patient's interpersonal relationships and the person's reactions and affects to and within those relationships holds the key to understanding the nature of the quality of the depression of BPD" [24, pp. 25].

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Associations between Identity Diffusion, Axis II Disorder, and Psychopathology in Inpatients with Borderline Personality Disorder

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Key Words
Affects · Borderline personality disorder · Depression · Identity diffusion · Mood · Psychopathology

Abstract
Background: Patients with borderline personality disorder (BPD) suffer from instability in their relationships, their affectivity, and their identity. However, the associations between these dimensions are not clear. The purpose of the present study was to investigate the relation between identity diffusion and psychopathology in BPD. ***Methods:*** In the second week of inpatient treatment, 52 patients with BPD were assessed with the Inventory of Personality Organization (IPO) and questionnaires measuring general psychiatric symptoms, mood states, and negative affects (SCL-90-R, BDI, STAI, and STAXI). A median split was examined to differentiate BPD patients with high identity diffusion from those with low identity diffusion. ***Results:*** BPD patients with high identity diffusion did not differ in their social data from BPD patients with low identity diffusion. However, BPD patients with high identity diffusion showed significantly higher levels of psychiatric symptoms, as well as higher anxiety, anger, and depression scores ($p < 0.01$). Moreover, they suffered more frequently from concurrent personality disorders ($p < 0.05$). ***Conclusions:*** These findings indicate an association of identity diffusion with psychopathological symptoms and features of personality disorder and emphasize the clinical significance of identity diffusion for patients with BPD.

Introduction

The prevalence of borderline personality disorder (BPD) is estimated to be between 1 and 2% for the general population, 10% for psychiatric outpatients, and 20% for psychiatric inpatients [1–4]. Psychiatric comorbidities are common in BPD, especially affective disorders, substance use disorders, and eating disorders [5–7]. Patients with BPD suffer from affective instability, interpersonal instability, and impulsivity [8, 9]. Affects such as anger and hostility and self-destructive behavior, as well as elevated stress and negative mood states, have been found [10–12].

Identity disturbance is one of the 9 diagnostic criteria of BPD [13]. It has been reported that this criterion has the most predictive power [14–16]. Moreover, the severity of identity disturbance and interpersonal problems has been found to be prognostic of the course of inpatient

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treatment over 6 months [17]. However, the findings on identity disturbance in BPD are inconsistent. Some studies have shown only moderate positive predictive power [18–20], while others have found a high negative predictive value and high sensitivity for the criterion identity disturbance [19, 21].

While the terms identity and identity disturbance were hardly used by S. Freud, they became a central part of psychoanalytical theory based on the identity concept of Erikson [22] and the borderline personality organization concept of Kernberg [23, 24].

The current psychological and sociological perspectives and conceptualizations of the term identity are manifold, e.g. (1) 'a subjective sense of personal sameness or continuity over time and across different situations and contexts', (2) an inner psychic structure of the mature personality, which (3) the individual has to engage in an ongoing struggle to realize and which occurs in processes of separation and individuation [15].

The psychodynamic perspective defines identity as the 'psychological structure that determines the dynamic organization of character' [25, p. 11]. This structure does not contain all of the different aspects of identity but – as an ego identity – it provides to some extent the basis for at least 3 further levels of identity such as personal identity, social identity, and collective identity. Ego identity manifests itself 'in conscious representations of the self, others, and the world in general, and in identifications with social groups, cultural norms, ideals, and values' [15, p. 346]. Thus, human identity can preliminarily be defined – to denote the aspect of subjectivity – as a sense of self or as the subjective experience of self, the feeling of the self's coherence, and its continuity over time and – to denote the diverse attributes – as a relatively coherent and stable center of behavior, of self-regulation, and of volitional acts, as an individual with stable and distinct traits, characteristics, needs, beliefs, values, and a unique (self-narrative) biography [15].

Identity diffusion is characterized by terms such as fragmentation, boundary confusion, and lack of cohesion in the subjective experience of self. It is distinguished from the typical identity crisis of adolescents in that adolescents, although involved in intense conflicts and confused about the attitudes of significant others to themselves, have a clear sense of the issues and their conflictual nature and are able to describe their own personality as well as the significant others with whom they enter into conflict in an appropriate, realistic, and integrated way. In contrast, patients with BPD demonstrate problems with internalized ethical values, norms, interests, and ideals and show a chaotic or contradictory attitude toward such value systems [23].

Thus, identity disturbance or identity diffusion is conceptualized by a lack of differentiated and integrated representations of the self and others, the lack of long-term goals, a negative self-image, and the lack of a sense of continuity in self-perception over time [14, 15, 22–24, 26–28].

As a result, patients experience rapid shifts in the way they view themselves and others, discontinuities and shifts of roles, and a sense of inner emptiness. Moreover, feelings of loss of integration and a sense of incoherence have been described [29, 30].

Fonagy et al. [31] pointed out the failure of BPD patients to develop the capacity to mentalize, i.e. to step into the mind of another and to imagine the way the other experiences the patient. Finally, Westen and Cohen [32] include in their definition of identity disturbance the above-mentioned attributes as well as others such as 'lack of a coherent life narrative or sense of continuity over time' or 'the loss of shared memories that help define the self over time' [32].

The paucity of empirical studies on identity and identity disturbance in BPD may stem partially from the difficulty of operationalizing and capturing the construct of identity disturbance in empirical clinical psychiatric research [33]. Studies in this area reported that: (1) less differentiated and integrated representations of the self and others were significantly related to the self-reported use of maladaptive strategies (e.g. self-injurious behavior) to regulate negative affective states [34]; (2) identity disturbance was found in half of the patients with personality disorders [35], and substantially higher incidences were found (e.g. 86% in the Risskov-I-study [36]); (3) in comparison with normal controls, contrasting attributes were identified more often in the self-description of patients with BPD [37]; (4) patients with BPD and severe identity disturbance showed a less favorable psychotherapeutic treatment outcome compared to those with less severe identity disturbance [17], and (5) in a factor analysis to ascertain whether identity disturbance is a unitary construct, Wilkinson-Ryan and Westen [38] distinguished 4 identity disturbance factors: role absorption, painful incoherence, inconsistency, and lack of commitment. All 4 factors, but particularly painful incoherence, distinguish patients with BPD from patients with a history of sexual abuse without BPD diagnosis.

It has been argued that identity diffusion may be correlated with high levels of self-rated psychopathology and negative affectivity [39].

The aim of this pilot study was to investigate associations of identity diffusion and psychopathology, such as general psychiatric symptoms, depression, anxiety, and anger.

Methods

Patients

Patients admitted consecutively to the Psychiatric Hospital of the University of Basel (Switzerland) and diagnosed as suffering from BPD according to DSM-IV-TR criteria were included in the study in the second week of treatment. Exclusion criteria were schizophrenia, schizoaffective disorder, active psychosis, and substance intoxication or substance withdrawal syndrome. IRB approval was obtained, and all patients gave their informed consent following a full explanation of the study.

Fifty-two patients diagnosed with BPD were included in this study. Out of the total sample, 42 (80.8%) patients were female and 10 (19.2%) male. The mean age was 29 years (SD 8.7).

Seventeen patients (32.7%) had only 1 personality disorder (BPD) without any other Axis II disorder, and only 2 BPD patients (3.9%) were without any comorbid Axis I disorder diagnosis. Table 1 lists the diagnostic characteristics of the sample.

Interviews

Clinically experienced psychologists interviewed patients who had previously screened positive for BPD using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P) [40] and DSM-IV Axis II Disorders (SCID-II) [41]. SCID-I and SCID-II are known for adequate-to-excellent internal consistency and interrater reliability [42, 43]. The interviewers had been trained to pay particular attention to distinguishing Axis I mental state conditions from Axis II personality trait phenomena.

Questionnaires

To assess identity diffusion, the Inventory of Personality Organization (IPO) [44, 45] was used. The scales of the IPO measure the constructs of identity diffusion, primitive defenses, reality testing, aggression, and moral values. Good validity and reliability have consistently been demonstrated for the IPO [46]. In order to generate subgroups for the severity of identity diffusion, a median split of the IPO scale identity diffusion was calculated.

The SCL-90-R [47] was administered to assess self-reported general psychiatric symptoms. The Beck Depression Inventory (BDI) [48], the Spielberger State and Trait Anxiety Inventory (STAI) [49], and the Spielberger State and Trait Anger Expression Inventory (STAXI) [50] were used to assess depression score, anxiety, and anger, respectively.

Statistical Analyses

All descriptive and inference-related statistical analyses were conducted with SPSS/15.0 for Windows. The parametric method used was the unpaired t test for group comparisons. χ^2 tests were used as nonparametric methods to test for intergroup differences. All statistical tests were considered significant at a 2-tailed level of $p < 0.05$.

Table 1. Clinical data of the sample

	Patients with BPD (n = 52)
Comorbid Axis II disorder (quantity), n (%)	
None	17 (32.7)
1–2	21 (40.4)
3–4	12 (23.1)
n/a	2 (3.9)
Comorbid Axis II disorder (cluster form), n (%)	
Cluster A	9 (17.3)
Cluster B	5 (9.2)
Cluster C	26 (50)
Comorbid Axis I disorder, n (%)	
None	2 (3.9)
Affective disorder	42 (80.8)
Anxiety disorder	27 (51.9)
Substance-related disorder	33 (63.5)
Eating disorder	19 (36.5)

Table 2. Social data of the sample

	High identity diffusion (n = 27)	Low identity diffusion (n = 25)	
Identity diffusion (mean ± SD)	68.6 ± 8.0	46.4 ± 10.6	t = –8.52 p < 0.0001
Age (mean ± SD), years	28.5 ± 8.0	29.4 ± 9.4	n.s.
Sex, n (%)			
Male	6 (22.2)	4 (16.0)	n.s.
Female	21 (77.8)	21 (84.0)	
Job situation, n (%)			
Employed	8 (29.6)	5 (20.0)	n.s.
Unemployed	19 (70.4)	20 (80.0)	
Family situation, n (%)			
Living with a partner	8 (29.6)	4 (16.0)	n.s.
Living alone	19 (70.4)	21 (84.0)	
Duration of BPD diagnosis, n (%)			
<1 year	–	3 (12.0)	n.s.
1–5 years	8 (29.6)	8 (32.0)	
>5 years	19 (70.4)	14 (56.0)	

Results

Social Data, Clinical Data, and Identity Diffusion

BPD patients with high identity diffusion and those with low identity diffusion did not differ significantly in their social data (table 2).

Table 3. Identity diffusion and negative affects

	High identity diffusion (n = 27)	Low identity diffusion (n = 25)	
IPO (mean ± SD)			
Primitive defenses	48.16 ± 5.76	35.28 ± 7.64	t = –6.90, p < 0.0001
Identity diffusion	68.60 ± 8.02	46.44 ± 10.64	t = –8.52, p < 0.0001
Reality testing	48.70 ± 12.46	32.36 ± 8.70	t = –5.44, p < 0.0001
Aggression	37.05 ± 7.91	28.25 ± 4.94	t = –4.77, p < 0.0001
Moral values	28.445 ± 7.03	21.00 ± 6.05	t = –4.08, p < 0.0001
SCL-90-R (mean ± SD)			
Global severity index	1.79 ± 0.58	0.98 ± 0.56	t = –5.16, p < 0.0001
BDI (mean ± SD)			
Depression score	29.26 ± 9.73	20.44 ± 11.29	t = –3.02, p = 0.004
STAI (mean ± SD)			
Trait anxiety	61.56 ± 8.42	50.38 ± 11.21	t = –4.08, p < 0.0001
STAI (mean ± SD)			
State anxiety	58.84 ± 11.55	48.67 ± 12.93	t = –2.99, p = 0.004
STAXI (mean ± SD)			
Trait anger	24.19 ± 7.11	18.04 ± 5.70	t = –3.43, p = 0.001
STAXI (mean ± SD)			
State anger	20.45 ± 8.68	14.40 ± 4.30	t = –3.22, p = 0.003

Identity Diffusion and Psychiatric Symptoms

Identity diffusion (IPO) and the self-rated psychiatric symptoms (SCL-90-R, BDI, STAI, and STAXI) correlated significantly with each other ($p < 0.01$). Table 3 gives an overview of the psychiatric symptoms of the groups with high and low identity diffusion. General psychiatric symptoms, depression scores, state anxiety, trait anxiety, state anger, and trait anger as well as all other IPO scales including primitive defenses, reality testing, aggression, and moral values were significantly higher in the group with high identity diffusion.

Identity Diffusion and Cooccurring Axis I and Axis II Diagnosis

While the groups did not differ with respect to concurrent Axis I disorder, the frequency of concurrent personality disorders was different in both groups. As shown in table 4, the group with high identity diffusion suffered more frequently from further personality disorders, mainly a concurrent cluster C personality disorder.

Discussion

Examination of identity diffusion and psychopathology has shown a significant association between identity diffusion, depression score, severity of psychiatric symptoms, and negative affects. Our findings support the results of a nonclinical sample study which has already shown a correlation between identity diffusion and negative affects [46], as well as the results of an interview study with BPD patients, measured with the Structured Interview of Personality Organization (STIPO), which found a lower level of personality structure including identity diffusion in the patients with more severe clinical symptoms [51].

The degree of identity diffusion could therefore be considered a dimension of BPD or an expression of clinical severity. Identity diffusion could be regarded as a specific stable feature of patients with BPD [52] or of patients with severe personality disorders in general [53] or, like other psychopathological items, as a feature of a severely psychopathological state.

Our results may represent 2 BPD groups with borderline pathologies of different severities. We found high values of all IPO scales and frequent additional Axis II disorders in the BPD group with high identity diffusion, which supports the concept of a borderline personality organization with more or less severe personality disorder [53]. Positive correlations between high identity diffusion, primitive defense mechanisms, interpersonal problems, impulsivity, and antisocial personality traits have also been also shown in previous studies [54]. Moreover, a BPD subgroup has been found to be characterized

Table 4. Identity diffusion and concurrent diagnosis

	High identity diffusion (n = 27)	Low identity diffusion (n = 25)	
Axis I disorder, n (%)			
None	1 (3.7)	1 (4.0)	n.s.
Affective disorder	24 (88.9)	18 (72.0)	n.s.
Anxiety disorder	15 (55.6)	12 (48.0)	n.s.
Substance use disorder	17 (63.0)	16 (64.0)	n.s.
Eating disorder	11 (40.7)	8 (32.0)	n.s.
Axis II disorder (cluster form), n (%)			
None	4 (14.8)	13 (52.0)	$\chi^2 = 8.37$, p = 0.04
Cluster A	5 (18.5)	4 (16.0)	n.s.
Cluster B	4 (14.8)	1 (4.0)	n.s.
Cluster C	18 (66.7)	8 (32.0)	$\chi^2 = 6.44$, p = 0.01
Axis II disorder (quantity), n (%)			
None	4 (14.8)	13 (52.0)	$\chi^2 = 8.37$, p = 0.04
1–2	12 (44.4)	9 (36.0)	n.s.
3–4	10 (37.1)	2 (8.0)	$\chi^2 = 6.17$, p = 0.013
n/a	1 (3.7)	1 (4.0)	n.s.
Axis II disorder (quantity), mean ± SD	2.00 ± 1.41	0.75 ± 0.99	Z = –3.26, p = 0.001

by elevated levels of identity diffusion, aggression, and antisocial features [55]. The interaction between identity diffusion and psychopathology remains unclear. Either identity diffusion predisposes to an exacerbation of psychopathological symptoms, or psychopathological states (e.g. emotional instability) cause the disintegration of the self and object representation. Empirical research on these interactions and possible causal relations remain to be carried out [51].

The concept of identity diffusion as a marker of a personality structure implies that differences in demographic data such as job situation and family situation are to be expected. Contrary to our expectations, we were not able to show a significant difference in social functioning between the 2 groups. The reason may be the small number of patients in each group.

In principle, our results suggest that the degree of identity diffusion presents a measurement of the severity of BPD or any other personality disorder, as proposed in the revision of the diagnostic categories for personality disorders in DSM-V and ICD-11 (www.dsm5.org). DSM-IV describes identity disturbance in BPD as being 'characterized by shifting goals, values, and vocational aspirations' [13, p. 651], underscoring commitment and social functioning as fundamental elements of the ego identity status [26, 56]. However, identity diffusion seems rather to manifest itself in specific fundamental factors such as 'painful incoherence' or 'inconsistency' [38] which distinguish BPD and other personality disorders from other types of psychopathologies. Regarding the specificity of the criterion, further research is needed to develop a differentiated and operationalized term for identity diffusion in order to avoid an equivocal concept. The detected correlation of identity diffusion with psychopathology and Axis-II comorbidity, respectively, could be interpreted in such a way that the degree of identity diffusion (as a result of the impaired development of a sense of self-identity) indicates a clinical measure for the severity of personality disorders which the recommendations for a revised definition of personality disorders in DSM-V intend to include [57].

Based on the assumption that the positive change in BPD symptoms manifests itself more in alterations in self-destructive behavior than in the personality traits which are close to temperament (e.g. impulsivity) [58, 59], the further course and the changes in both dimensions – i.e. identity diffusion and psychopathology – need to be examined. At present, there are no empirical findings that sufficiently explain the associations between the distinct core symptoms of BPD [60].

Further research should focus on the clarification of an empirically operationalized concept of identity diffusion and investigate the clinical relevance of a thorough personality structure level in determining the severity of

a personality disorder. Regarding prognosis, important therapeutic indications (e.g. expected hospitalization) or possible therapeutic strategies hinge on determining the severity of a personality disorder.

Our study contains several methodological limitations. Because of the small sample sizes in each group, the generalizability of these results is limited. Our failure to find significant differences in social data between BPD patients with low and high identity diffusion should be interpreted with great care. Further research is needed to investigate identity diffusion in the different types of personality disorders and to examine the question of whether identity diffusion changes in the natural course of the personality disorder.

In conclusion, the associations of negative affects, mood, and identity diffusion indicate the clinical importance of identity diffusion in patients with BPD.

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Change in Identity Diffusion and Psychopathology in a Specialized Inpatient Treatment for Borderline Personality Disorder

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Objectives: Patients with borderline personality disorder (BPD) show various psychopathological symptoms and suffer especially from disturbance in their identity. The purpose of the study was to investigate changes—particularly in affective BPD symptoms and identity diffusion—during a structured, disorder-specific inpatient treatment (DST) that combined a psychodynamic transference-focused psychotherapy approach with modules of dialectical behavioural skills training.
Method: In a prospective, two-group comparison trial, 44 patients with BPD were assessed with questionnaires addressing identity diffusion and state, as well as trait affective psychopathology, before and after 12 weeks of inpatient treatment. Thirty-two patients received DST, whereas 12 patients were given inpatient treatment-as-usual (TAU). The patients were allocated in a non-random procedure for two groups, in order of admission and availability of treatment options in the DST unit.
Results: In the pre-post-comparison, the DST group showed a significant decrease in identity diffusion ($p < 0.001$) and improvements in instability of the image of self and others ($p < 0.008$), as well as in pathological (trait and state) symptoms. However, there was no significant improvement in the TAU group.
Conclusions: After a 12-week inpatient treatment, the findings indicate significant improvements in the DST group in typical affective borderline symptomatology and in the personality structure feature of identity diffusion. This highlights the significance of a short-term specific inpatient therapy for BPD.

Key Practitioner Message:

- A structured, disorder-specific inpatient treatment of patients diagnosed with borderline personality disorder (BPD) combined a psychodynamic transference-focused psychotherapy treatment approach (focusing on pathological features in personality organization, particularly on non-integrated images of self and others) with modules of dialectical behavioural skills training. This treatment is associated with a decrease in identity diffusion of these patients after 12 weeks of treatment.
- The treatment is also related to a significant decrease in borderline typical psychopathological symptoms such as depressive symptoms, as well as an improvement in state anger.
- The outcomes of this structured, disorder-specific inpatient treatment of severely ill BPD patients indicated the relevance of intensive short-term inpatient psychotherapy in terms of psychopathological improvements as well as initial changes in structural personality organization.

Keywords: Borderline Personality Disorder, Identity Diffusion, Inpatient Treatment, Disorder-Specific Treatment, Transference-Focused Psychotherapy

OBJECTIVES[1]

Borderline personality disorder (BPD) is a serious psychiatric problem. The mean prevalence is 1.35% in the

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general population (Torgersen, Kringlen, & Cramer, 2001), 15–25% amongst psychiatric inpatients (Gunderson, 2009; Torgersen, 2005) and 50% amongst psychiatric inpatients with personality disorders (PDs) (Lenzenweger, 2008; Lenzenweger, Lane, Loranger, & Kesserl, 2007; Lenzenweger, Loranger, Korfine, & Neff, 1997; Widiger & Weissman, 1991). Symptoms of negative affectivity and affect dysregulation, impulsivity and instability in interpersonal relationships characterize the borderline syndrome (Gunderson & Links, 2008; Sanislow, Grilo, & McGlashan, 2000). Affects of anger and hostility, self-destructive behaviour and elevated stress and negative mood states are characteristic (Henry *et al*., 2001; Walter *et al*., 2008; Zanarini *et al*., 1998). There is a high comorbidity in BPD with other Axis II PDs, as well as Axis I disorders (depression, anxiety, eating disorders, post-traumatic stress disorder and substance use disorder) (Leichsenring, Leibing, Kruse, New, & Leweke, 2011; Walter *et al*., 2009; Zanarini *et al*., 1999).

Personality disorders are associated with distorted and maladaptive thinking about oneself and others. The components that substantially constitute personality functioning are identity, self-direction, empathy and intimacy (Bender, Morey, & Skodol, 2011; Morey *et al*., 2011).

The revision of DSM-5 admittedly maintains a categorical model for the diagnosis of PDs but includes a new trait-specific methodology in a separate part of Section III to encourage further research in this area (APA, 2013). It takes into account dimensions of *self-functioning* and *interpersonal functioning*, particularly impairments in identity or in self-directedness (instability in goals, aspirations, values etc.). However, despite its important role, research on problems of identity in BPD is still sparse (Brazier *et al*., 2006; Levy *et al*., 2006).

Kernberg *et al*. focussed on this diagnostic core concept of 'identity' with regard to PDs, stressing a notion of identity that 'provides a psychological structure determining the dynamic organisation of character' (Clarkin, Yeomans, & Kernberg, 2006, p. 11). On the basis of his psychodynamic theory of personality, Kernberg defines identity diffusion as 'a structural, pathological consolidation of the internalised world of object relations' (Kernberg, 2006, p. 980), which is reflected in disordered and unintegrated representations, both of the self and of other people. Many researchers and clinicians consider identity diffusion or disturbance to be one of the core diagnostic criteria for BPD (Jørgensen, 2006, 2010; Jørgensen *et al*., 2009). Kernberg further argues that identity diffusion is 'the key anchoring point of the differential diagnosis of milder types of character pathology and neurotic personality organisation, on the one hand, and severe character pathology and borderline personality on the other' (Kernberg, 2004, p. 61).

As we reported previously (Sollberger *et al*., 2012), the paucity and inconsistencies of the results of empirical studies on identity and identity diffusion may stem partially by the difficulty in operationalization, accompanied by confusion of the constructs of identity diffusion in empirical clinical psychiatric research (Oldham *et al*., 1985).

Several operationalized and evidence-based disorder-specific outpatient treatments have proven to be effective in terms of symptom decline and improvements in general functioning (Sollberger & Walter, 2010; Stoffers *et al*., 2012; Yeomans, Levy, & Meehan, 2012). Whereas dialectical behavioural therapy (DBT) focuses primarily on an improvement in the fundamental affect dysregulation (Lynch, Chapman, Rosenthal, Kuo, & Linehan, 2006) of BPD patients, the psychodynamic approach of transference-focused psychotherapy (TFP) (Clarkin *et al*., 2006) addresses—besides general and specific symptomatology—structural features of personality, particularly the core pathological feature of identity diffusion. At its root, identity diffusion, as the lack of a coherent, integrated sense of self and others, characterizes the pathological personality organization of a broad range of severe PDs organized at the borderline level, including BPD; it is treated in TFP as it manifests in therapeutic transference, i.e., as a form of interpersonal manifestation of intrapsychic conflicts amongst different internal states.

The efficacy of both the operationalized treatment of DBT (Stoffers *et al*., 2012) and that of TFP (Clarkin, Levy, Lenzenweger, & Kernberg, 2007; Doering *et al*., 2010; Giesen-Bloo *et al*., 2006) has been evaluated in several randomized controlled trials.

Regarding the current state of research, it is striking that only a few studies have been published on inpatient treatments (Bartak *et al*., 2011; Bohus *et al*., 2013) with even fewer investigating the combination of different and specific therapeutic approaches in the treatment of BPD patients.

In the recent Cochrane Review on psychological therapies for patients with BPD (Stoffers *et al*., 2012), all but two studies—one is unpublished, and the other was excluded from the review for lack of randomization (Bohus *et al*., 2004)—were conducted in an outpatient setting. Participants in the Bateman and Fonagy (1999) study were partially hospitalized. The study of Steil, Dyer, Priebe, Kleindienst, and Bohus (2010, the unpublished study in the review) included several stages—from a diagnostic outpatient phase to an inpatient stay and an additional outpatient booster session after the end of the inpatient treatment. But the main interventions were conducted in an inpatient setting. A systematic review of 11 studies of DBT in inpatient treatment of BPD (Bloom, Woodward, Susmaras, & Pantalone, 2012) shows considerable variation in treatment configuration and duration. However, the findings suggest that DBT may be effective in reducing symptoms related to BPD in inpatient settings. In an uncontrolled inpatient study using psychoanalytic-interactional psychotherapy, Leichsenring, Masuhr, Jaeger, Dally, and Streeck (2010) report improvements with large effect sizes in target and general symptoms, interpersonal problems and contentedness of life of patients with BPD.

In many countries (Germany, Netherlands, Switzerland etc.), intensive inpatient treatment of borderline patients or patients with other severe PDs is still very common and can probably facilitate subsequent outpatient psychotherapy (Bohus *et al*., 2004; Stoffers *et al*., 2012). Indications for inpatient treatment are (acute or chronic) suicidality, severe or risky impulsive behaviour, problems with current substance abuse, major psychosocial problems or a negative therapeutic reaction that severely impedes outpatient psychotherapy. Inpatient treatment particularly emphasizes the potential risks of non-specific inpatient treatment (regression, severe enactments etc.) (Dammann, 2012; Zeeck *et al*., 2009). As an overall goal, inpatient treatment seeks to be effective in reducing psychopathological symptoms related to BPD. It also aims to initiate a change in features of pathological personality organization such as identity diffusion. TFP specifically addresses the latter as manifestation of a split mental structure.

To summarize, even though identity diffusion is thought to be a core feature of BPD that is addressed by TFP, changes in identity diffusion as an effect of specific psychotherapeutic interventions have hardly been examined. Only a few studies have reported improvements in personality functioning, such as personality organization (Doering *et al*., 2010) or reflecting functioning and narrative coherence (Levy *et al*., 2006). Moreover, despite recent studies that examined the effectiveness of inpatient treatment of BPD (Bartak *et al*., 2011; Bohus *et al*., 2013; see also Bloom *et al*., 2012), there is still a need of studies that particularly prove clinical approaches under natural conditions in treatment programmes that combine different evidence-based psychotherapeutic therapies.

Therefore, this study sought to examine the effectiveness of a structured, disorder-specific inpatient treatment (DST) of BPD patients in a specialized disorder-specific psychiatric inpatient unit. This unit employs the viable clinical approach of combining psychodynamic treatment with modules of dialectical behavioural skills training (distress tolerance, emotional regulation, interpersonal effectiveness and mindfulness). More precisely, we focussed on the characterological core feature of identity diffusion in BPD patients and investigated changes in identity diffusion during the DST in comparison with inpatient treatment-as-usual (TAU). We argued that BPD patients in the DST setting not only show a decrease in symptomatology but—in contrast to the TAU group—also exhibit less identity diffusion after the psychotherapeutic treatment.

METHODS

Study Design and Participants

Patients participated in an inpatient study for BPD patients (Basel Borderline Inpatient Study BABIS). The aim of this study was to compare the effects of DST versus TAU.

The study was designed as a prospective, non-randomized, two-group comparison inpatient study for patients with a main diagnosis of BPD. Patients who had been admitted consecutively to the Psychiatric University Hospital Basel (Switzerland), had been diagnosed as suffering from BPD by trained and experienced clinicians according to the DSM-IV-TR criteria and were between the ages of 18 and 65 years were included in the study. Exclusion criteria were schizophrenia, schizoaffective disorder and bipolar disorder. Moreover, patients with intoxications and current drug or alcohol abuse as well as with mental retardation were excluded, whereas patients with a remitted substance use disorder were included.

The local ethics committee (EKBB Basel) obtained IRB approval, and all patients signed written informed consent, following a full explanation of the study.

Sampling

At baseline, 60 patients (37 DST/23 TAU) were interviewed. Five patients (three DST/two TAU) failed to complete the questionnaire and were excluded from the study. Fifty-five patients were allocated in two parallel groups due the clinical procedure described below. Forty-four patients (32 DST/12 TAU) completed the second part of our study after 12 weeks.

Our sample consisted of 44 patients diagnosed with BPD. Of the total sample, 35 (79.5%) patients were female, and nine (20.5%) were male. The mean age was 29.6 years (standard deviation = 9.2). Fifteen BPD patients (34%) had one PD, 18 (41%) had two or three PD diagnoses and 11 patients (25%) had more than three PD diagnoses. Additionally, 38 patients suffered from concurrent major depressive disorder (86.3%), 27 had a substance use disorder (61.4%), 24 had an anxiety disorder (54.5%) and 17 BPD patients had a concurrent eating disorder (38.6%), according to the SCID-I/P (First, Spitzer, Gibbon, & William, 1996).

The study was designed to contain two parallel groups in two different wards with different therapists, one with DST and one with TAU. Until recently, the inpatient unit providing DST in the Psychiatric University Hospital, Basel, was the only one in the region. It is dimensioned for 14 patients. Because of this DST treatment facility shortage, many of the borderline patients entering the psychiatric hospital for psychotherapeutic treatment had to be referred to another inpatient unit providing TAU. At the same time, patients from other parts of Switzerland were referred specifically to the DST unit, due to the conditions of payment specified by their insurances. Therefore, randomization was not feasible in this clinical trial. Entry into the treated DST group followed the order of admission and availability of treatment options in the DST unit.

Timing

The assessment point for both DST and TAU was the first week after entering the clinic. Post-testing was conducted 12 weeks after the initial assessment.

Settings and Treatments

Borderline personality disorder patients were assigned to a DST that combines a psychodynamic TFP approach with modules of dialectical behavioural skills training in the psychotherapy ward of the Psychiatric University Hospital or to TAU in a general psychiatric inpatient ward of the same hospital.

Treatment-as-usual consisted of clinical management (supportive treatment, social psychiatry and psychopharmacotherapy) from the psychiatric services. Patients in this group generally attended one non-specific psychotherapeutic session per week with a psychiatrist, psychoeducation in group therapy, supportive talks with staff nurses and one session per week with a social worker. Once a week, the senior physician of the unit supervised the staff team. All team members were experienced in treating patients with BPDs but not trained in specialized evidence-based treatments for BPD.

Disorder-specific inpatient treatment combined twice-weekly individual TFP sessions (in accordance with the TFP treatment manual; see Clarkin *et al*., 2006) with a primary therapist trained in TFP, together with twice-weekly TFP-oriented psychodynamic group therapy with nurses and a social worker (similar to TAU sessions), as well as weekly supervision and consultation meetings for the therapists. In addition and depending on the demonstrated improvements in affect regulation (Linehan *et al*., 2006), patients in the DST unit attended weekly DBT-based skills-training groups conducted by trained staff nurses to augment the TFP treatment. As already mentioned above, this specific inpatient treatment unit combined psychodynamic treatment with modules of dialectical behavioural skills training. Whereas DBT skills sessions focus particularly on mindfulness and on coping with extreme affect states and dysfunctional behaviour, TFP targets the conflicts amongst the patient's internal representations of self and others within the transference and interpersonal problems (Yeomans *et al*., 2012).

Disorder-specific inpatient treatment was performed in a specialized psychotherapeutic unit with a set stay length of 12 weeks, whereas TAU was provided with variable lengths of stay (mean 11.33 weeks). Thus, treatment dose (Howard, Kopta, Krause, & Orlinsky, 1986) expressed as period of treatment was comparable in both groups but slightly in favour of DST.

Psychopharmacologically experienced psychiatrists on every unit prescribed the medication. All patients in both groups were on medication deemed appropriate by the psychiatrists and in accordance with the recommended APA guidelines (Soloff, 2000).

Interviews

Clinically experienced interviewers were trained for structured clinical interviewing. To determine Axis I and Axis II disorders, they interviewed subjects screened in the clinical enrolment interview by experienced medical doctors as being positive for BPD. These determinations were made on the basis of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P) (First *et al*., 1996) and for DSM-IV Axis II disorders (SCID-II) (First, Gibbon, Spitzer, Williams, & Benjamin, 1997).

Outcome Measures and Questionnaires

We chose identity diffusion as a primary outcome measure because this complex construct of Kernberg's object relations model of BPD, which underlies our study, is thought to be the core feature of borderline personality organization.

Changes in affective psychopathology (i.e., anxiety, aggression and depression) were chosen as a secondary outcome measure because of their relevance to emotional and affective state and trait instability, from which BPD patients suffer.

To assess identity diffusion before and after treatment, an instrument based on Kernberg's concept of borderline personality organization was used: the Inventory of Personality Organisation (IPO) (Clarkin, Foelsch, & Kernberg, 2000; Dammann, Smole-Lindinger, & Buchheim, 2002; Lenzenweger, Clarkin, Kernberg, & Foelsch, 2001). This inventory measures the theoretically based constructs of identity diffusion, primitive defences, reality testing, aggression and moral values. We also used a recently published analysis of the empirically examined factor structure of the primary scales for IPO (Ellison & Levy, 2012). In contrast to the three theoretical constructs of the IPO defined by Kernberg (coherence of identity, maturity of defences and capacity for reality testing), Ellison and Levy identified a four-factor structure from an empirical analysis (performed on a large, albeit non-clinical, sample [$n = 1260$] and using exploratory structural equation modelling as well as multiple regression). The structure includes the following: (1) 'Instability of Self and Others'; (2) 'Instability of Goals'; (3) 'Psychosis'; and (4) 'Instability of Behaviour'. The authors argue that factors 1 and 2 'can be seen as separate aspects of the identity diffusion construct' with regard to both the pattern of factor loadings and the relationships to external measures of self-concept stability. Moreover, the first factor seems to be a 'general factor' because it contains far more items than the three others. We therefore calculated our data on this empirically evaluated basis, too.

To assess the affective state and trait symptoms, as well as changes in comorbidity of depression, we applied the Beck Depression Inventory (BDI) (Hautzinger, Bailer, Worrall, & Keller, 2000), the State-Trait Anxiety Inventory (Laux, Glanzmann, Schaffner, & Spielberger, 1981) and the State-Trait Anger Expression Inventory (Schwenkmezger, Hodapp, & Spielberger, 1992).

Good validity and reliability have consistently been demonstrated for the IPO (Lenzenweger *et al*., 2001), as well as for the other, above-mentioned self-rating instruments (Barnes, Harp, & Jung, 2002; Dozois, Dobson, & Ahnberg, 1998; Forgays, Forgays, & Spielberger, 1997; Mueller, Bongart, Heiligtag, & Hodapp, 2001).

Drop-Out Rates

Eleven patients (20%; two DST/nine TAU) failed to complete the second part of our study for several reasons: three patients (one DST/two TAU) left the clinic early, four patients (one DST/three TAU) no longer wished to participate and four patients (four TAU) could not be contacted.

Statistical Analyses

All statistical analyses were conducted with SPSS/20.0. Assumptions of homoscedasticity and tests for normal distribution were performed prior to the analysis. χ^2-tests were used for testing intergroup differences in nominal and ordinal scales. Pre-post-comparison was analysed with the paired *t*-test for normally distributed and the Wilcoxon signed rank test for non-normally distributed data. The unpaired *t*-test and the Mann–Whitney *U*-test were performed for group comparison (corresponding to the distribution of the data). All statistical tests were considered significant at a paired level of $p < 0.05$.

RESULTS

Social and Clinical Characteristics

Table 1 shows severe BPD psychopathology with frequent Axis I and Axis II pathologies at baseline. Because of our recruitment process that was characterized by natural clinical

Table 1. Social and clinical data of the sample

	DST ($n = 32$)	TAU ($n = 12$)	p
Age, mean (SD)	26.7 (6.5)	29.4 (9.4)	$t = -4.14$, $p = 0.001$
Sex, *n* (%)			
Male	6 (18.8)	3 (25.0)	ns
Female	26 (81.2)	9 (75.0)	
Job situation, *n* (%)			
Employed and partially employed	12 (37.5)	4 (33.3)	ns
Apprenticeship	8 (25.0)	0 (0.0)	ns
Unemployed	7 (21.8)	2 (16.7)	ns
Disability pension	6 (18.8)	10 (83.3)	$t = 3.989$, $p = 0.001$
Family situation, *n* (%)			
Living with a partner	7 (21.9)	1 (8.3)	ns
Living alone	25 (78.1)	11 (91.7)	
Duration of the illness, *n* (%)			
<1 year	2 (6.3)	1 (8.3)	ns
1–5 years	13 (40.6)	3 (25.0)	
>5 years	17 (53.1)	8 (66.7)	
Axis I disorder, *n* (%)			
None	2 (6.3)	0 (0.0)	ns
Affective disorder	27 (84.4)	11 (91.7)	ns
Anxiety disorder	19 (59.4)	5 (41.7)	ns
Substance-related disorder	18 (56.3)	9 (75.0)	ns
Eating disorder	13 (40.6)	4 (33.3)	ns
Axis II disorderCluster form, *n* (%)			
None	9 (28.2)	6 (50.0)	ns
Cluster A	6 (18.8)	1 (8.3)	ns
Cluster B	2 (6.3)	2 (16.7)	ns
Cluster C	21 (65.6)	3 (25.0)	$\chi^2 = 5.8$, $p = 0.02$

BPD = borderline personality disorder. DST = disorder-specific treatment. TAU = treatment-as-usual. SD = standard deviation. ns = non-significant.

conditions, we had significant differences between the two treatment groups in the following three variables: comorbidity in PDs of the C type (anxious and fearful disorders), age and disability pensions. On average, BPD patients in the DST group were 3 years younger than those in the TAU group. A total of 83.3% ($n = 10$) of the TAU group had a full disability pension, in contrast to 18.8% ($n = 6$) in the DST group ($p = 0.001$). However, despite this difference, the study participants in both groups were fully or partially employed, and therefore, no significant between-group difference has been found concerning the employment status.

Psychopathology and Identity Diffusion at Baseline

As shown in Table 2, the groups did not differ at baseline in terms of psychopathological affective features (state and trait anger and anxiety, depressive symptoms). In contrast, both identity diffusion—one of the theoretical constructs of the IPO (Clarkin *et al*., 2000)—and instability of the sense of self and of others—an empirical factor of the IPO (Ellison & Levy, 2012)—differed significantly between DST and TAU (Table 2). Moreover, there was also a significant group difference in terms of primitive defences (IPO)

Changes in Psychopathology and in Self-Functioning

After the DST group's 12 weeks of inpatient treatment, we found significant changes in the theoretical construct of identity diffusion (IPO), as well as in the empirical factor 'instability of self/others' (IPO empirically evaluated). In contrast, identity diffusion and instability of the sense of self and of others increased in the TAU group but not significantly. Thus, the significant group difference at baseline in this regard was absent at the second measuring point. The other parameters of personality organization features showed no significant differences after treatment for either group (Table 3). In addition, a significant decrease in affective psychopathology was measured in patients participating in the DST. Thus, these patients showed a significant improvement in self-reported depression—even though the depressive symptoms still indicated clinically relevant depression after inpatient treatment. Furthermore, improvements in state anger and a tendency towards improvement in anxiety states were demonstrated. However, no differences were found for the TAU group.

DISCUSSION AND CONCLUSIONS

Identity Diffusion

Our combined TFP-DBT, disorder-specific, structured inpatient treatment of patients diagnosed with BPD focused on both the decline in psychopathological symptoms and on an improvement in structural features of personality organization, particularly changes in identity diffusion.

Table 2. Between-group comparisons at pre-treatment

	DST ($n = 32$)	TAU ($n = 12$)	p
IPO (theoretical construct), mean (SD)			
Identity diffusion	62.71 (12.81)	47.75 (13.74)	$t = 3.70$, $p = 0.001$
Primitive defences	43.42 (8.94)	37.41 (9.08)	$t = 2.19$, $p = 0.034$
Reality testing	41.19 (12.67)	38.83 (16.66)	ns
Aggression	34.11 (8.21)	32.27 (7.12)	ns
Moral values	25.59 (7.80)	21.83 (5.94)	ns
IPO (empirically evaluated four-factor structure)*, mean (SD)			
Instability in self and others	94.41 (17.95)	75.33 (19.90)	$t = 3.31$ $p = 0.002$
Instability in goals	5.61 (2.38)	4.75 (2.65)	ns
Psychosis	21.42 (8.65)	20.67 (9.70)	ns
Instability in behaviour	19.10 (5.56)	16.40 (6.81)	ns
STAXI, mean (SD)			
State anger	18.56 (8.07)	15.00 (6.83)	Z = -2.12, p = 0.034
Trait anger	22.10 (7.30)	18.67 (6.27)	ns
STAI, mean (SD)			
State anxiety	56.25 (11.0)	50.70 (14.86)	ns
Trait anxiety	58.33 (8.65)	53.58 (14.22)	ns
BDI, mean (SD)			
Depression score	27.16 (9.16)	23.64 (12.92)	ns

*Ellison & Levy, 2012

DST = disorder-specific treatment. TAU = treatment as usual. IPO = Inventory of Personality Organisation. STAXI = State-Trait Anger Expression Inventory. STAI = State-Trait Anxiety Inventory. SD = standard deviation. ns = non-significant.

Table 3. Pre-post-tests of disorder-specific inpatient treatment ($n = 32$) and treatment-as-usual ($n = 12$) groups

Variable	Mpre	SDpre	Mpost	SDpost	*t*	*p*
IPO (theoretical construct)						
Identity diffusion—DST	62.71	12.81	58.49	14.01	2.95	0.006
Identity diffusion—TAU	47.75	13.74	48.92	12.28	−0.38	0.711
Primitive defences—DST	43.42	8.94	41.89	10.63	1.24	0.225
Primitive defences—TAU	37.41	9.08	36.92	7.96	0.27	0.789
Reality testing—DST	41.19	12.67	39.99	13.98	0.43	0.673
Reality testing—TAU	38.83	16.66	37.70	12.21	0.42	0.683
Aggression—DST	34.11	8.21	33.12	7.95	0.98	0.337
Aggression—TAU	32.27	7.12	29.88	7.67	2.15	0.060
Moral values—DST	25.59	7.80	24.67	6.98	0.93	0.358
Moral values—TAU	21.83	5.94	20.83	5.88	0.73	0.481
IPO (empirically evaluated four-factor structure)*						
Instability in self/others—DST	94.41	17.95	88.35	21.61	2.85	0.008
Instability in self/others—TAU	75.33	19.90	77.35	17.80	0.46	0.964
Instability in goals—DST	5.61	2.38	5.27	2.62	1.07	0.293
Instability in goals—TAU	4.75	2.65	5.82	2.69	−1.70	0.120
Psychosis—DST	21.42	8.65	21.11	9.28	0.20	0.840
Psychosis—TAU	20.67	9.70	19.54	7.45	1.16	0.274
Instability in behaviour—DST	19.10	5.56	18.99	5.65	−0.22	0.832
Instability in behaviour—TAU	16.40	6.81	15.91	6.24	1.27	0.233
BDI						
BDI—DST	27.16	9.16	21.39	12.31	3.65	0.002
BDI—TAU	23.64	12.92	18.86	14.49	0.94	0.370
STAI						
State anxiety—DST	56.25	11.00	53.10	12.86	1.36	0.184
State anxiety—TAU	50.70	14.86	49.33	14.03	0.41	0.690
STAXI						
State anger—DST	18.56	8.07	14.28	5.26	−3.18	0.001
State anger—TAU	15.00	6.83	17.18	8.38	−1.67	0.126

*Ellison & Levy, 2012

IPO = Inventory of Personality Organisation. DST = disorder-specific treatment. TAU = treatment as usual. BDI = Beck Depression Inventory. STAI = State-Trait Anxiety Inventory. STAXI = State-Trait Anger Expression Inventory. M = mean. SD = standard deviation.

Transference-focused psychotherapy for patients with BPD targeted identity diffusion (Levy *et al*., 2006), i.e., the non-integrated, split-affect (sequestered in all negative, all positive) images of self and others. It specifically addressed conflicts amongst the patients' internal affective states, focusing on the transference in the therapeutic relation. With regard to these affective states—which are often barely tolerated—the DBT's teaching of skills helped the patient regulate and tolerate impulses and overwhelming affects. Thus, the psychotherapeutic work within the firm and structured inpatient treatment frame focused primarily on the activated and affect-related patient–therapist dyads in clarifying, confronting and interpreting the affective states in their relation to images of self and others. This approach was intended to achieve a decline in symptoms. It was also intended to reduce identity diffusion and to initialise a process of integration. This process was to be continued in an outpatient setting.

Our results demonstrate a significant decrease in identity diffusion and instability of the image of self and others during the relatively short, specific inpatient treatment, compared with inpatient TAU. Although the DST group showed a significant higher Axis II Cluster C comorbidity, which presumably impedes a successful outcome (Zanarini *et al*., 2004), these patients benefited from inpatient psychotherapy. It seems that the absence of the TFP-based emphasis on identity diffusion in the TAU group explains its lack of positive change.

One objection might be that the effect is due to the level of severity, which differs at baseline between the two groups in terms of Axis II Cluster C comorbidity, as well as in terms of identity diffusion (measured by the IPO) (for a similar discussion regarding self-injury, see Doering *et al*., 2010). But it also might be true that particularly Cluster C comorbidity with (neurotic) traits of anxiety and avoidance leads patients in the DST group—besides their additional aggression—to more intense use of the therapeutic relationship and to more space for quiet observation, reflection and description of their own emotional states underlying the therapeutic dyads. This might

be a benefit in terms of prognosis, since it is of particular concern with respect to splitting mechanisms in self-image and object representations. As reported previously, borderline patients show a poorly integrated image of the self and others, in contrast to controls with major depressive disorders (Dammann *et al*., 2011): they predominantly display an altruistic, superficial and suffering self-image and see aggressive tendencies only in other persons. The TFP-based DST focuses precisely on such split, polar-opposite affect dispositions that influence partial representations and are supposed to disrupt patients' capacity for integrating their partial representations of self and others (Kernberg, 2006). However, bearing in mind the severity of the disorder, the significant improvement in identity diffusion in a relatively short DST inpatient treatment, which focuses on achieving structural or personality change, indicates the effectiveness and the necessity of the treatment.

Moreover, the significant pre-treatment group difference in the level of identity diffusion makes it methodologically difficult to appropriately attribute the positive outcome effects in identity diffusion in the DST group—in contrast to the TAU group—to the specific therapeutic intervention. The effect could be due to the methodological possibility of 'regression to the mean'. Thus, for purely technical reasons, patients with higher identity diffusion at baseline in the DST group might show significant improvements in contrast to patients with less identity diffusion in the TAU group. However, relative to the TAU group, the DST group also exhibited improvements in the other outcome measures (depression and state anger). There, it appears at least probable that the effect on identity diffusion is specifically due to the intervention, namely, the TFP in DST that focuses particularly on this feature. That provides evidence that DST might be a treatment of special benefit to patients with severe BPD pathology. Furthermore, the other significant pre-treatment group differences should be taken into account when interpreting the superiority of DST inpatient treatment with regard to the improvement in identity diffusion: the younger age of the DST group and the greater number of disability pensions in the TAU group (Table 1). Both may be related to the chronicity of the disorder, as the BPD patients (with a mean age of around 30 years) are more frequently on disability pensions due to their illness and are only partially employed, whereas the younger patients in the DST group often are still in an apprenticeship or fully employed. Therefore, younger patients have more optimistic social perspectives than the elder patients. However, in the natural course of BPD, as Zanarini and her group showed (Zanarini, Frankenburg, Hennen, & Silk, 2003), there is a general decline in the course of ageing. Thus, elder patients in our TAU group might benefit from the small positive difference in age relative to the DST group. On the basis of our data, it is difficult to define the relevance of these factors, as it is not clear which group was in better initial condition. The DST group had the advantage of fewer disability pensions, which may indicate less advanced illness. However, the group also had the disadvantages of greater Cluster C comorbidity, higher state anger and more primitive defence mechanisms.

Disorder-specific treatment, particularly the TFP items that address identity diffusion, seems to be able to change identity diffusion after intensive inpatient treatment for a limited period. Admittedly, more research is needed, particularly regarding the long-term outcome.

Affective Psychopathology: Anger, Anxiety and Depression

In addition, the DST group showed a significant decrease relative to the TAU group in state anger but not in anxiety. The latter might be due to Cluster C comorbidity, given that pervasive feelings of social inhibition and inadequacy do not change in the socially intense environment of the unit and in the short term. The significant decrease in state anger in DST patients apparently indicates the beginning of reorganization in perceiving and experiencing others in interaction. These findings are totally consistent with those of Clarkin *et al*. (2007), who showed a significant reduction in anger as a specific improvement of the TFP outpatient treatment, in contrast to the lack of improvement in the dialectical behaviour treatment. This was also similar to the results reported by Bohus *et al*. (2004), where the DBT group showed significant improvement in every outcome measure except anger. Aggression in terms of state anger is one of the psychotherapeutic core features during inpatient treatment: during the intense social coexistence in the unit, aggressive interactions are focused both in the therapeutic milieu in the ward and in the psychotherapeutic group and individual therapy. Generally, patients promptly become confronted with their aggressive behaviour by fellow patients or care workers and therapists. However, changes in trait anger, as well as state and trait anxiety, will last much longer than 12 weeks and require a longer treatment phase, generally in an outpatient setting.

In agreement with Bateman and Fonagy (1999), who reported significant group differences in the improvements in depression in BPD outpatients in mentalization-based therapy, we found a significant decrease in depressive symptoms in the DST group. This contrasts with the majority of RCTs of specific BPD outpatient treatments, where no group differences in depression were reported (Clarkin *et al*., 2007; Doering *et al*., 2010; Linehan, Armstrong, Suarez, Allmon, & Heard, 1991; Linehan *et al*., 2006; Verheul *et al*., 2003). Our findings cannot be attributed to a high severity of depressive illness as Doering *et al*. (2010) do for the Bateman and Fonagy study, in which patients started with a BDI score of 36.0. Our

patients started with a BDI score of 27.2 in the DST group (comparable with 25.8 for the patients in the Doering study), and there was no significant group difference at baseline. Thus, it is more likely to be a specific effect of the intense and, in contrast to TAU, highly structured inpatient treatment of the specialized unit. During DST, it may be that the modifications in the conceptions of self and others in interaction, particularly affect-mobilizing therapies (such as TFP-oriented group and individual therapy combined with DBT-based mindfulness therapy, as provided by the psychotherapeutic treatment programme), affect changes in depressive syndrome by improving the capacity to observe, reflect and describe emotional states. Clarification processes in group and individual psychotherapy and TFP typical reflections on changing dyads in transference and its unconscious motives (Levy *et al*., 2006), as well as a safe and stable therapeutic environment, might be relevant in this regard.

Strength and Limitations

The strength of the study is its natural clinical design. Moreover, it is one of still few studies that investigate DST of severe BPD patients, whereas most findings concerning the psychotherapeutic outcome of these patients are based on outpatient treatments (see Cochrane Review, Stoffers *et al*., 2012). In addition, the study profits from recent findings on the empirically evaluated IPO (Ellison & Levy, 2012) with the core parameter of our study: identity diffusion and instability in self and others measured by the IPO. The results therefore provide an important empirical basis.

The study contains several methodological limitations. First of all, randomization was not feasible for practical reasons, as we stated above (see section on 'Sampling'). In addition, the study has a small sample size, particularly in the TAU group, because patients either let themselves be put on a waiting list instead of an inpatient treatment as usual in another, unspecialized psychiatric ward or they utilized only a short time intervention of a few weeks. Moreover, there was a higher drop-out rate in the TAU group than in the DST group (two DST, nine TAU) indicating a stronger therapeutic alliance in the DST group. Furthermore, there are significant group differences at baseline, which makes it difficult to appropriately weigh the relevance of these factors in terms of the outcome results. Because of these differences as well as of the possibility of further unobserved differences due to non-randomization, the generalizability of the results is limited.

In addition, in their inpatient setting, TAU group patients received a lower treatment dose on average. This lower dose was due to differences both in the length of stay between various units and their clinical programmes and the number of therapeutic contacts per week. In some cases, it was also because of early discharge from inpatient treatment. Thus, the interpretations of the positive treatment results have to be qualified by these limitations. This has to be carefully taken into account. Our results need to be confirmed in further research. Also because of the lack of matching in the allocation method of the present study, a larger study sample is needed in order to control the diverse treatment methods.

CONCLUSION

In conclusion, there are significant improvements in identity diffusion as well as in affective psychopathology under a disorder-specific, TFP-DBT combined inpatient treatment over 12 weeks. In spite of the substantial methodological limitations of the study, the results indicate that disorders-specific inpatient treatment may be effective.

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The Impact of Interpersonal Problems in Borderline Personality Disorder Inpatients on Psychopathology and Treatment Outcome

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Abstract

Background: Borderline Personality Disorder (BPD) is a very common illness. Interpersonal problems are one of the core features of the disorder next to affective instability and identity disturbance. The purpose of the study was to investigate the impact of the severity of interpersonal problems in borderline inpatients on borderline features and psychopathology over time.

Methods: 37 inpatients with BPD were assessed with the Structured Clinical Interviews for DSM-IV Axis I and II Disorders (SCID I, SCID II) in their first week in the clinic and additionally had to complete a questionnaire (IIP-C, BDI, STAI, STAXI, SCL-90-R). After 12 weeks of transference focused based disorder specific treatment patients had to fill out the same questionnaire once more. Since 7 patients didn't complete the two questionnaires only 30 patients were included in the study. In order to generate two subgroups for the severity of interpersonal problems, a median split of the IIP general scale was calculated.

Results: Patients with higher interpersonal problems showed significant higher scores in identity diffusion and psychopathological symptoms but not in anger or impulsivity. After treatment, all patients showed a significant decrease in identity diffusion, depression, state anger and most interpersonal subscales. If compared separately, patients with higher interpersonal problems showed a significant decrease in identity diffusion, state anger, general interpersonal problems and several interpersonal subscales (non-assertive, exploitable, overly nurturant). In contrast patients with lower interpersonal problems at baseline showed a significant decline in depression and trait anger.

Conclusions:

Higher interpersonal problems are connected with other borderline features and psychopathological symptoms but not anger and impulsivity. After 12 weeks treatment the findings indicate significant improvements in typical borderline features for the group with higher interpersonal problems. It seems that high interpersonal problems are a positive predictor for psychodynamic disorder specific treatment for BPD.

Keywords: borderline personality disorder, interpersonal problems, TFP, treatment outcome

Introduction

Borderline is one of the most common personality disorders that affects about 0.5 to 5,9% of the general population, around 10% psychiatric outpatients and 15-20% psychiatric inpatients. (Lenzenweger et al., 2007; Trull et al., 2010). The main characteristics include interpersonal problems, affective instability, disturbed identity as well as impulsivity.

In DSM-V (APA, 2013), interpersonal problems are described as a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation. As well as frantic efforts to avoid real or imagined abandonment. The new DSM-5 section III model of personality disorders (APA, 2013) defines, in agreement with Otto F. Kernberg's theory of structural personality organization, interpersonal functioning as the capacities for empathy and intimacy, with four severity levels of dysfunction.

Among patients with BPD, associations have been found between BPD symptoms and interpersonal sensitivity, interpersonal ambivalence, interpersonal aggression, need for social approval, and lack of sociability (Stepp et al., 2011). Interpersonal dysfunctioning in BPD patients appears to be one of the best discriminator for diagnosis (Modestin 1987; Zanarini et al., 1990; Gunderson, 2007).

Impairment of this social interaction is consistent with clinical experience and the literature (Stepp et al., 2009; Salzer et al., 2013). Although individuals with BPD may report a general tendency to exhibit interpersonal passivity, their behavioral repertoires can include more assertive approaches to engaging the social environment (Russell al., 2007). Physiological reactivity may be related to interpersonal ambivalence in BPD features (Dixon-Gordon et al., 2013).

In the study of Wright et al. (2013a) BPD symptom counts were unrelated to the primary dimensions of the IIPC, but were related to generalized interpersonal distress. In their study a latent class analysis revealed six homogeneous interpersonal classes with prototypical profiles associated with Intrusive, Vindictive, Avoidant, Nonassertive, and moderate and severe Exploitable interpersonal problems. These classes differed in clinically relevant features (e.g., antisocial behaviors, self-injury, past suicide attempts). A German study with over 200 BPD inpatients (Salzer et al., 2013) showed that the interpersonal style of BPD had a significant influence on interpersonal distress and global severity symptoms.

Interpersonal problems are often a central focus of treatment and endure even after other symptoms have remitted (Clarkin, Yeomans & Kernberg, 1999; Zanarini et al., 2007) In contrast with the stability of interpersonal styles in other personality disorders, the stability of the interpersonal patterns in BPD patients is not clear.
Wright and colleagues (2013b) investigated the stability of interpersonal problems in BPD patients over the course of a year and found that „interpersonal dysfunction on borderline pathology is stable in its severity but unstable in the style of its manifestation“ (pp. 1094).

Aims of the study

The aim of our study is to closer examine the role of interpersonal problems in borderline personality disorder patients. In a first step we want to examine whether the severity of interpersonal problems is correlated with other borderline or psychopathological features. Another point is the question whether interpersonal problems diminish during treatment and if the degree of interpersonal problems has an impact on treatment results.

Methods

Study Design and Participants
All Patients were inpatients at the Psychiatric Hospital of the University of Basel and were diagnosed with a borderline personality disorder (BPD) according the DSM-IV-TR criteria. Patients participated in an inpatient study for BPD patients (Basel Borderline Inpatient Study (BABIS)) and were treated at a specialized psychotherapeutic unit with a set stay length of 12 weeks. They received a disorder specific treatment (DST) that combines a psychodynamic TFP approach with modules of dialectical behavioural skills training. Aims of the BABIS were to compare the effects of this specialised treatment versus treatment as usual and to identify the possible influence of subgroups within the heterogeneous group of BPD patients Detailed descriptions of the aims, methods and sample characteristics of the Basel Borderline Inpatient Study (BABIS) supported by a research grant from the Swiss National Science Foundation have been reported separately (Agarwalla et al., 2013).
Exclusion criteria were schizophrenia, schizoaffective disorder, active psychosis or acute manic episode. Written informed consent was obtained from each patient. The study was approved by the local Ethics Committee (EKBB).
The assessment point was the first week after entering the clinic. Post-testing was conducted 12 weeks after the initial assessment.

Interviews

Clinically experienced interviewers attended a special education of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P) (First et al., 1996) and for DSM-IV Axis II Disorders (SCID-II) (First et al., 1997) and were trained to pay particular attention to distinguishing Axis I mental state conditions from Axis II personality trait phenomena. The SCID I and II are semi-structured interviews for assessing clinical and personality disorders. High interrater reliability has been shown for both interviews (Lobbestael et al., 2011; Maffei et al., 1997).

Questionnaire data

For evaluation of interpersonal criteria we used the Inventory of Interpersonal Problems (Horowitz et al., 2000), a 64-item self-report instrument designed to measure interpersonal deficiencies and excesses in 8 subscales (e.g. too responsible, too controlling) and a general scale. The IIP-C bases upon a two-dimensional circular model of interpersonal disfunctioning with two axis (Domineering vs. Nonassertive and Overly nurturant vs. Cold). The validity and reliability of the IIP-C scales have been demonstrated (Horowitz et al., 2000).

In order to generate subgroups for the severity of interpersonal problems, a median split of the IIP general scale was calculated.

To measure the general psychiatric symptoms and subjective complaints, we administered the SCL-90-R (Franke, 1995), the Beck Depression Inventory (Hautzinger et al., 2000), the Spielberger State and Trait Inventory (Laux et al., 1981), and the Spielberger State and Trait Anger Inventory (Schwenkmezger et al., 1992).

Statistical Analyses

All statistical analyses were conducted with SPSS/20.0. Assumption of homoscedasticity and normality distribution was checked prior to the analysis. χ^2-tests were used for testing intergroup differences. Comparison of the two groups was conducted with Student's t-Test for normally distributed data and Mann Whitney U-Test if a normal distribution wasn't given. For comparison before and after treatment, we ran a paired t-Test (normal distributed data) and Wilcoxon signed rank (no normal distribution assumed) respectively. All test results were considered significant at a two-sided level of $p< 0.05$.

Results

37 patients diagnosed with borderline personality disorder (BPD) were included in the study and interviewed. 5 patients didn't complete the questionnaire and were therefore excluded. 2 patients didn't finish the second part of our study and were also counted out. Of the 30 patients included in the study, 25 (83.3%) were female, 5 (16.7%) male. The mean age was 26.4 years (SD = 6.1) (see Table 1).

28 patients (93.3%) were diagnosed with a comorbid Axis I Disorder, most frequently with an affective disorder (*n*=25, 83.3%); An anxiety disorder was diagnosed in 18 patients (60%). 22 patients (73.3%) showed a comorbid Axis II disorder, predominant a Cluster C disorder (*n*=20, 66.7%).

Table 1 :
Demographic and Clinical Characteristics of Borderline Patients

	Borderline patients *(*n*=30)*
Age, mean (SD)	26.4 (6.1)
Gender, n *(%)*	
Male	5 (16.7)
Female	25 (83.3)
Family situation, n *(%)*	
Living alone	23 (76.7)
Living with a partner	7 (23.3)
Current employment, n *(%)*	
Employed (full/part time) /Apprenticeship	18 (60)
Unemployed/disability pension	12 (40)
Duration of illness, n (%*)*	
<1 year	1 (3.3)
1 year to 5 years	13 (43.3)
5 to 10 years	4 (13.3)
10 to 20 years	8 (26.7)
> 20 years	4 (13.3)
Comorbid Axis I disorder, n (%*)*	
None	2 (6.7)
Affective disorder	25 (83.3)
Anxiety disorder	18 (60)
Substance related disorder	17 (56.7)
Eating Disorders	13 (43.3)
Comorbid Axis II disorder, n (%*)*	
None	8 (26.7)
Cluster A	6 (20)
Cluster B	2 (6.7)
Cluster C	20 (66.7)

Notes. SD= standard deviation

Interpersonal Problems

A median split of the IIP Total item score was conducted to generate two severity groups. Since the mean over all patients of this score was rather high (1.84 compared to 1.28 in the

general population [Horowitz et al., 2000]) we refer to the two groups as one with higher interpersonal problems (HI) and one with lower interpersonal problems (LI). The HI-group had an IIP Total item mean score of 2.25 compared to 1.47 for the LI-group.

Between-group comparisons at pre-treatment

Patients with higher interpersonal problems showed significant higher scores in identity diffusion and several psychopathological symptoms (e.g. depression, anxiety, GSI) but not in anger or impulsivity (see Table 2) . They also were more often diagnosed with a comorbid Cluster C disorder and had more years of education. Regarding Interpersonal subscales, the HI group showed higher data in all scales; the scores were significant except for the scales Domineering/Controlling and Vindictive/Self-Centered.

Table 2 :
Comparison of Higher and Lower Interpersonal problems

	*Higher interpersonal problems (*n*=15)*	*Lower interpersonal problems (*n*=15)*	
IIP, mean (*SD)*			
Total Score	2.25 (0.24)	1.47 (0.32)	t= -7.58, p=0.000**
Domineering/Controlling	7.00 (5.40)	5.60 (3.20)	n.s.
Vindictive/Self-Centered	10.88 (5.80)	9.53 (4.19)	n.s.
Cold/Distant	15.07 (5.27)	10.87 (5.02)	t= -2.23, p=0.034*
Socially Inhibited	21.67 (4.61)	15.00 (6.21)	t= -3.34, p=0.002**
Nonassertive	24.27 (6.05)	13.60 (6.69)	t= -4.58, p=0.000**
Overly Accommodating	23.20 (4.54)	12.73 (4.48)	t= -6.36, p=0.000**
Self-Sacrificing	25.93 (3.60)	16.31 (4.63)	t= -6.35, p=0.000**
Intrusive/Needy	16.27 (4.13)	10.68 (4.47)	t= -3.56, p=0.001**
IPO, mean (*SD)*			
Primitive defenses	45.20 (7.97)	40.92 (9.33)	n.s.
Identity diffusion	67.27 (11.45)	57.87 (12.89)	t= -2.11, p=0.044*
Reality testing	43.20 (13.74)	38.87 (11.75)	n.s.
Aggression	33.96 (9.02)	33.67 (7.42)	n.s.
Moral values	24.60 (7.40)	25.80 (7.95)	n.s.
SCL-90-R, mean *(SD)*			
Global severity index	1.83 (0.50)	1.14 (0.48)	t= -3.85, p=0.001**
BDI, mean *(SD)*			
Depression Score	30.98 (7.22)	23.89 (9.70)	t= -2.27, p=0.032*
STAI, mean *(SD)*			
State Anxiety	62.00 (9.02)	52.27 (9.53)	t= -2.87, p=0.008**
Trait Anxiety	62.93 (7.58)	55.17 (7.05)	t= -2.90, p=0.007**
STAXI, mean *(SD)*			
State Anger	17.34 (6.15)	19.19 (8.67)	n.s.
Trait Anger	20.60 (7.39)	23.67 (7.51)	n.s.
Cluster C Disorder, n (%)			
Yes	13 (86.7)	7 (46.7)	χ^2= 5.40, p=0.020*
No	2 (13.3)	8 (53.3)	

*Notes. SD= standard deviation, ns= non-significant, * p<0.05, ** p<0.01*

Pre-post-tests of all patients

After treatment, all patients show a significant decrease in various variables. There is a significant change in all IIP subscales except for the scales Domineering/Controlling, Cold/Distant and Intrusive/Needy (see Table 3). Other significant decreases are IPO identity diffusion, BDI as well as in State Anger. The scores decline in all areas except for the IIP scale Domineering/Controlling.

Table 3:
Pre-post-tests of all patients

Variable	*Mpre*	*SDpre*	*Mpot*	*SDpost*	*t*	*p*
IIP						
Total Score	1.86	0.48	1.66	0.47	3.10	0.004**
Domineering/Controlling	6.30	4.42	7.03	4.92	-1.21	0.235
Vindictive/Self-Centered	10.21	5.02	8.67	4.69	2.13	0.042*
Cold/Distant	12.97	5.49	12.20	5.01	0.78	0.443
Socially Inhibited	18.33	6.35	16.21	5.49	2.26	0.031*
Nonassertive	18.93	8.29	16.60	7.93	3.30	0.003**
Overly Accommodating	17.97	6.93	15.73	6.82	2.35	0.026*
Self-sacrificing	21.12	6.37	17.75	5.93	4.46	0.000**
Intrusive/Needy	13.47	5.09	12.23	4.56	1.84	0.077
IPO						
Identity diffusion	62.57	12.90	58.27	14.20	2.91	0.007**
Primitive Defenses	43.06	8.80	41.82	10.81	1.00	0.324
Reality testing-	40.41	12.51	39.51	13.97	0.63	0.536
Aggression	33.81	8.12	33.09	8.08	0.71	0.481
Moral values	25.20	7.58	24.46	6.70	0.75	0.461
BDI						
Depression Score	27.44	9.15	21.68	12.21	3.46	0.002**
STAI						
State Anxiety	57.14	10.38	54.01	12.36	1.26	0.216
Trait Anxiety	59.05	8.20	57.34	9.79	1.29	0.204
STAXI						
State Anger	18.27	7.45	14.53	5.34	3.35	0.002**
Trait Anger	22.13	7.48	20.42	6.36	1.94	0.062
SCL-90						
Global Severity Index	1.48	0.60	1.37	0.67	1.16	0.255

*Notes. M= Mean, SD= standard deviation, * p<0.05, ** p<0.01*

Pre-post tests conducted for both groups separately showed some differences: Patients with higher interpersonal problems showed a significant decrease in identiy diffusion, state anger and the IIP general scale as well as several subscales (nonassertive, exploitable, overly nurturant). On the other hand, patients with lower interpersonal problems showed a significant decline in depression, trait anger and only the interpersonal subscale overly nurturant.

Discussion

Although the relevance of interpersonal dysfunction of BPD patients is obvious, the underlying mechanisms are not clear.

Interpersonal problems could be associated with attachment styles (Bornstein et al., 2010; Gunderson & Lyons-Ruth, 2008; Levy, 2005; Agrawal, Gunderson et al., 2004, higher vulnerable narcissism (sensitivity), rejection hypersensitivity, problems with intimacy, fear of abandonment (Gunderson & Lyons-Ruth, 2008; Ayduk et al., 2008; Lejuez et al., 2003), disturbed empathy or mentalization, impaired social cognition, low trust or higher degree of temperamental aggressiveness or anger (Critchfield, Clarkin, Levy, & Kernberg, 2008; McCloskey et al., 2009).

Despite the limited number of participants of the study, reducing the generalisability of the results, two main conclusions seem possible. More psychopathology or comorbidity is not always an indicator for worse treatability. To the contrary specific symptomatologies can be indicators for better prognosis and less comorbidities can show less therapeutic effects (see also the results of Bateman & Fonagy, 2013).

Our results are especially important as persisting severe impairments in general, social and interpersonal dysfunction have been shown in longitudinal studies for Borderline Personality Disorder (Zanarini et al., 2007; Zanarini et al., 2012; Gunderson et al., 2011). In the study of Choi-Kain et al. (2010) demandingness and boundary violations turned out to remit quickly while affectively oriented interpersonal features related to intolerance of loneliness and conflicts about dependency persisted. The quality of current relationships of patients with BPD predicted the outcome at a 2-year-follow-up (Gunderson et al., 2006).

Low interpersonal functioning cannot be sufficiently explained by affective dysregulation theory (Jeung & Herpertz, 2014, p. 221). Interpersonal problems and identity diffusion (lack of a stable sense of one's self) seem to be related.

In general psychiatric hospitals hospitalized BPD patients feel hostilely repulsed by staff members and affronted by other patients (Benjamin & Wonderlich, 1994). The relationship of BPD patients interpersonal problem styles and alliance are not clear (Salzer et al., 2013).

The results of our study are congruent with prior results that transference-focused psychotherapy could have specific advantages, compared with other treatments, in the interpersonal field (f.e. reflective functioning) (Levy et al. 2006) and that changes in identity diffusion, pseudoaltruistic interpersonal features (Non-assertive, Overly acommodating, Self-Sacrificing) and associated anger are possible and crucial.

Intensive and disorder-specific psychodynamic psychotherapy may have an especially favorable outcome for Borderline Personality Disorders with severe interpersonal problems, due to the fact that transference-focused psychotherapy is focusing on interpersonal difficulties.

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